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TABLE OF CONTENTS FIRST PAGE

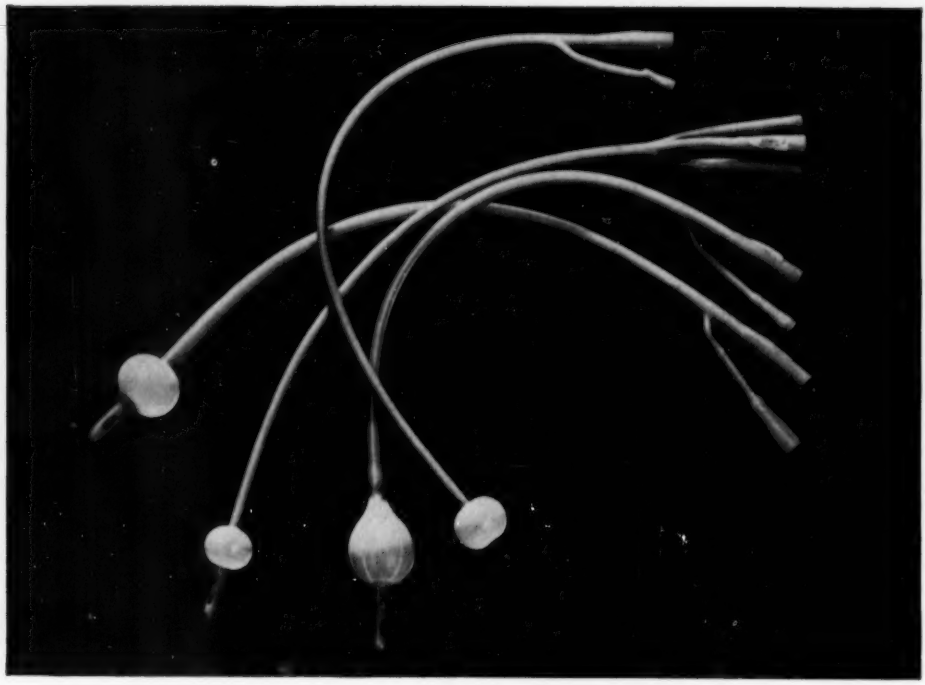

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TABLE OF CONTENTS

EDITORIALS

Present Status of the Treatment of Carcinoma of the Esophagus PAGE
Richard H. Sweet, M.D. 1

ORIGINAL ARTICLES

Surgical Reversal of Superior Vena Cava Syndrome
B. A. McArt, M.D.; F. B. Ramsey, M.D.; W. A. Tosick, M.D., and K. R. Woolling, M.D., M.S. (Med.), Indianapolis 4

Successful Treatment of Cardiac Arrest
Bernard L. Brofman, M.D., and Juan J. Perez-Alvarez, M.D., Cleveland 12

Acute Necrotizing Gastritis
Albert Behrend, M.D.; Albert B. Katz, M.D., and John W. Robertson, M.D., Philadelphia 18

Basal Cell Carcinoma of the Anus
Allen P. Klippel, M.D., and William L. Tomlinson, M.D., St. Louis 25

Acute Intestinal Obstruction Secondary to Meckel's Diverticula
Donald P. Chance, M.D.; Robert T. Hood Jr., M.D., and John M. Waugh, M.D., Rochester, Minn. ... 28

Pitfalls, Precautions, and Complications in Cardiac Resuscitation
Hugh E. Stephenson Jr., M.D., Columbia, Mo.; L. Corsan Reid, M.D., and J. William Hinton, M.D., New York 37

Surgical Treatment of the Auriculotemporal Syndrome
K. B. Coldwater, Ph.D., M.D., St. Louis 54

Studies of the Aged
James Mithoefer, M.D., and John C. Mithoefer, M.D., Cooperstown, N. Y. 58

Lymphomas of Spinal Epidural Space
J. Grafton Love, M.D.; Ross H. Miller, M.D., and James W. Kernohan, M.D., Rochester, Minn. ... 66

Bile Peritonitis Due to Spontaneous Perforation of the Common Duct
Harold J. Muendel, M.D., Basking Ridge, N. J., and Robert H. Null, M.D., Plainfield, N. J. 77

Retroperitoneal Duodenal Rupture Due to Blunt Trauma
Luther M. Keith Jr., M.D., Columbus, Ohio, and Buford H. Burch, M.D., Galveston, Texas 81

Clinical Experience in Intravenous Administration of Coconut Water
B. Eiseman, M.D., Denver; R. E. Lozano, M.D., Tela, Honduras, and T. Hager, St. Louis 87

Comparative Report of Partial Gastrectomy and Vagotomy in Treatment of Peptic Ulcer
Vladimir Jakovljevic, M.D., and Dejan Aleksic, M.D., Novi Sad, Yugoslavia 94

Preserving Arterial Segments for the Blood Vessel Bank
John M. Hammer, M.D.; Patrick H. Seay, Ph.D.; Albert De Groat, M.D., Kalamazoo, Mich., and Frank W. Prust, M.D., Detroit 97

Pancreatic Cystogastrostomy, a Definitive Treatment for Pseudocysts of the Pancreas
C. Richard Bowers, M.D., Dallas, Texas 101

TRENDS IN ORTHOPEDIC SURGERY

Marshall R. Urist, M.D., Los Angeles 113
Alvin J. Ingram, M.D., Memphis, Tenn. 119

CASE REPORTS

Retroperitoneal Metastatic Disease Due to Carcinoma of the Prostate
Charles D. Bonner, M.D., and Melvin Lyons, M.D., Boston 122

Strangulated Diaphragmatic Hernia with Gangrene of Stomach
William Hoffman, M.D.; Morris L. Levy, M.D.; Eugene Sole, M.D., and Alexander Lewitan, M.D., Brooklyn 125

Persistent Appendiceal-Sigmoid Fistula
W. Andrew Dale, M.D., Rochester, N. Y. 132

Special Notice 134

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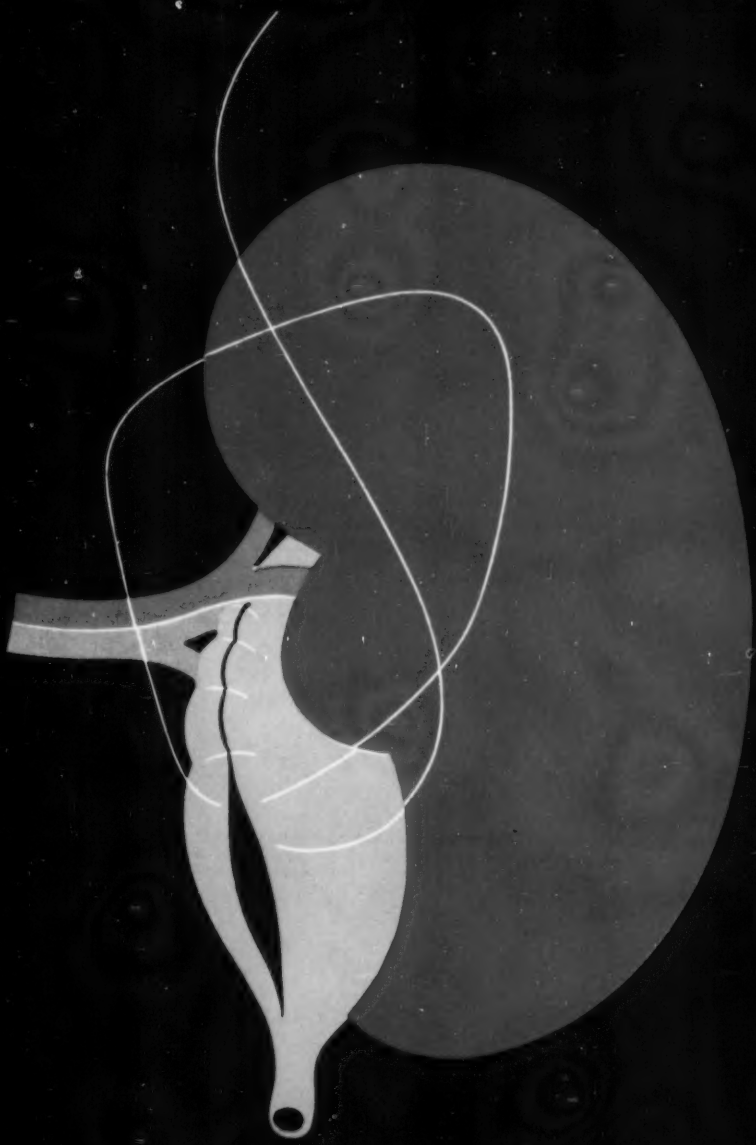

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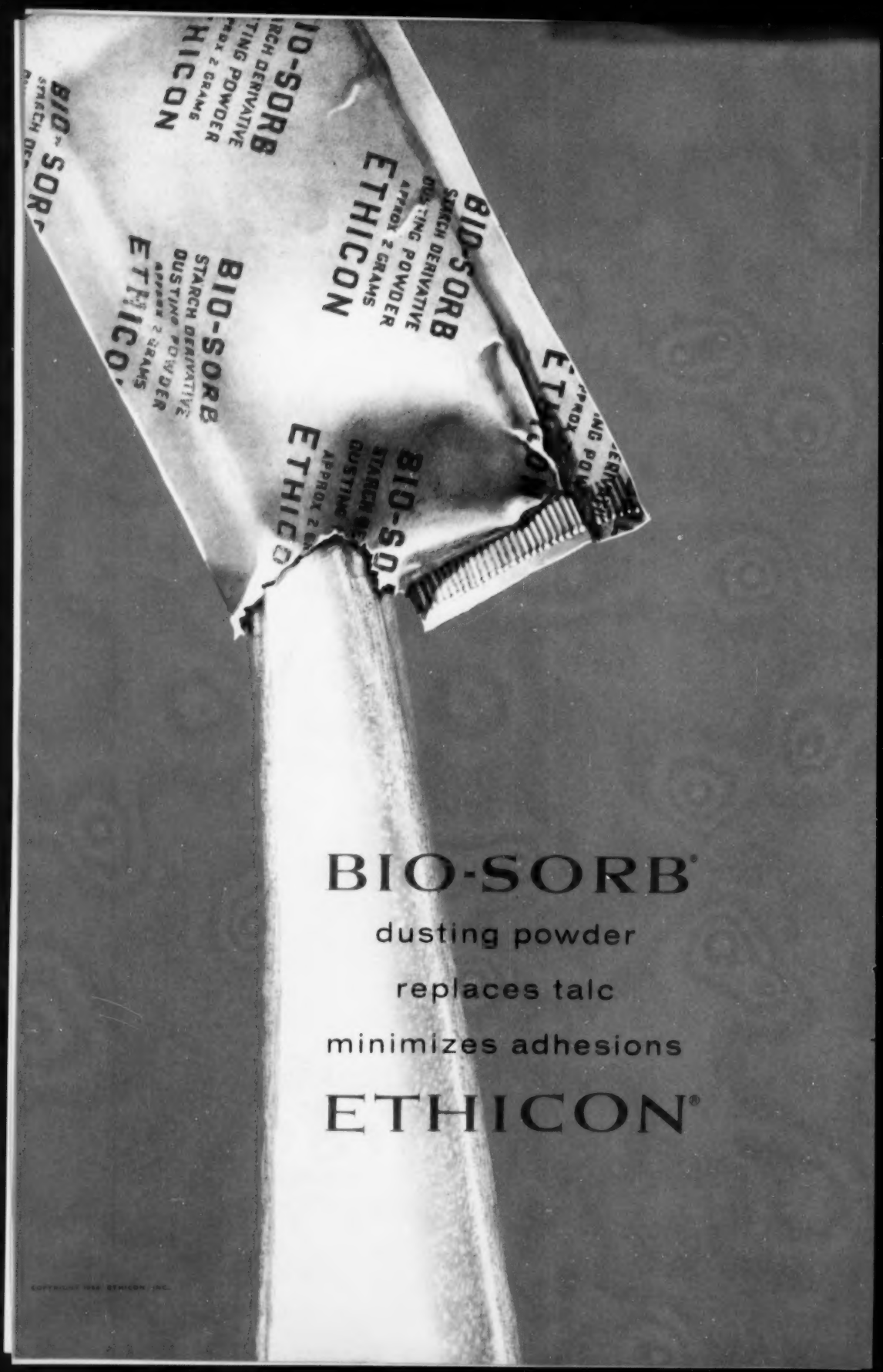
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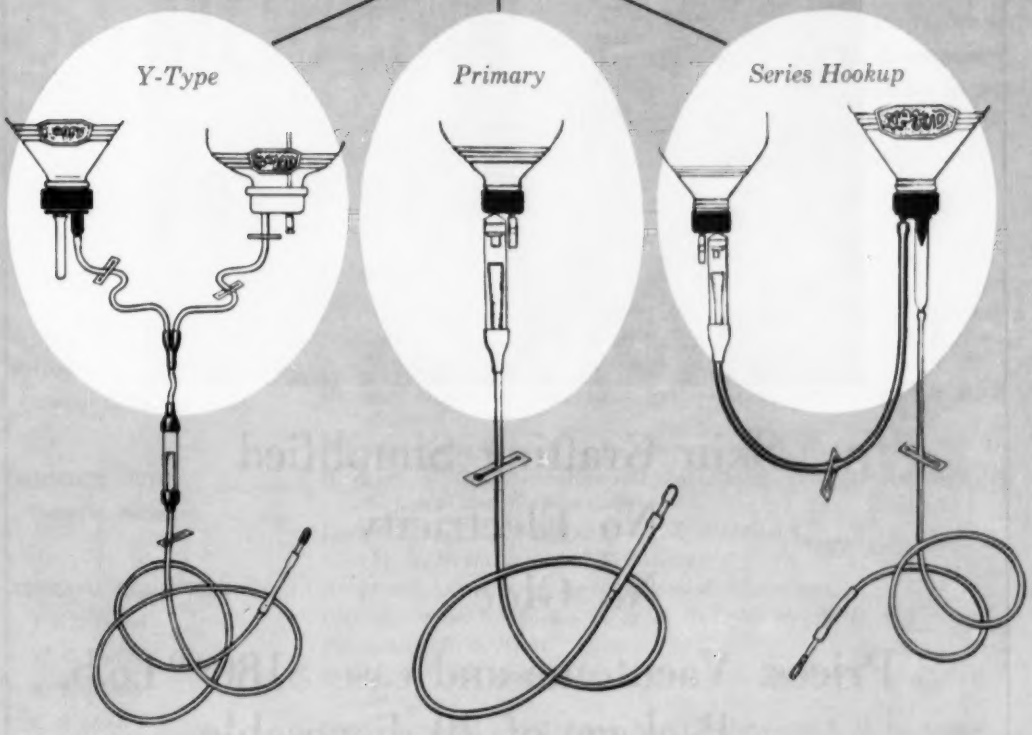

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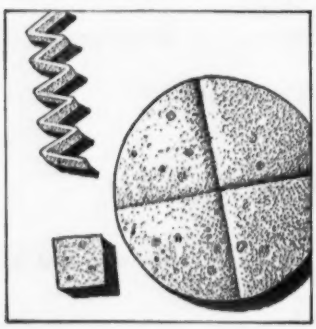

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VOLUME 69 JULY 1954 NUMBER 1

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Editorials

PRESENT STATUS OF THE TREATMENT OF CARCINOMA OF THE ESOPHAGUS

PERUSAL of the recent surgical literature and conversations with various surgeons reveal that, in this country particularly, there is an increasing sense of discouragement with the results of surgical treatment of carcinoma of the esophagus. This sometimes arises from the fact that premature conclusions are drawn from an inadequate experience. Obviously the law of statistical averages makes it possible for a small series to include by chance alone an unusually large number of either unfavorable or favorable cases. Thus among recent reports based upon a limited experience one author denies that there are any five-year survivals after surgical extirpation of the disease and another proclaims that in a group of patients operated upon for carcinoma of the lower esophagus, 41.6 per cent lived five years. Neither of these conclusions could be accurate because of the small size of the series in each instance.

Another reason for the current discouragement, but one which is difficult to evaluate without intimate knowledge, may be the technical inadequacy of the procedure carried out. It need not be emphasized that, as with carcinoma elsewhere, the operation for the removal of a carcinoma of the esophagus must include the extirpation of the local growth with a wide margin of normal tissue beyond it and also an extensive excision of the surrounding tissues and regional lymph nodes. To be sure, the technical difficulties in the way of accomplishing the ideal operation in some instances are actually insurmountable, but in any large series of cases there is an appreciable number with a favorable prospect.

In the present state of uncertainty as to the ultimate results of surgical treatment, it is not to be wondered that serious consideration is being given to the possible benefits to be obtained from roentgen-ray therapy in cases of carcinoma of the esophagus. As a matter of fact, Buschke and Cantril have recently expressed enthusiasm regarding the results obtained by supervoltage roentgen therapy.[1] Worth-while palliation was obtained in the majority of their patients. They contend furthermore that surgery offers no superiority from the point of view of length of survival. Their series includes 19 patients treated five or more years before evaluation, but only 14 had what they describe as complete treatment which may be considered comparable, so far as the efficacy of the method is concerned, to the surgical group in whom all the growth was apparently removed. There were three patients in their series alive at the end of five years (21 per cent of those receiving "complete treatment"). This series is too small, however, to be statistically significant.

In evaluating surgical extirpation of carcinoma of the esophagus it is now well established that, depending upon the topographical anatomy of the region involved, the adequacy of the operation from the point of view of cancer surgery increases from above downward as the growth may be found nearer and nearer to the cardia. Thus in the author's series taking into consideration all cases, whether favorable for removal or not, there were 159 patients who had been operated upon five or more years before the evaluation of the result and who survived the procedure. Among these the five-year survival rate is zero for those with tumors primary in the cervical segment, 4 per cent for those in the upper area, and 17 per cent for those in the low thoracic area and cardia.[2] With regard to the latter group it is interesting that the five-year survival rate is exactly the same for those with an epidermoid lesion as for those with an adenocarcinoma. These results are almost identical with those reported recently by Nakayama.[3]

It should be pointed out also that considering the nature of the disease and the age and infirmity of many of the patients, the current operative mortality of 7 per cent in the low group and 24 per cent in the higher cases is not prohibitive.

The author's experience is based upon the policy established at the outset (fifteen years ago) of offering the benefit of surgical extirpation to all patients whose growth could be removed, whether or not the disease appeared to be favorable for complete excision of the primary lesion and the regional lymph nodes. However, if the patients are grouped according to whether the tumor appeared to be favorable or unfavorable from the above viewpoint and the five-year survival results are then evaluated, the outlook is actually encouraging.

It can be demonstrated, for example, that in the surgical group of patients whose growth was favorable for removal and who were operated upon five or more years before evaluation, there were 64 who survived operation and 17 alive after five years (27 per cent of those who had adequate treatment). If these cases are analyzed further, it is found that among those with a lesion at the cardia or in the lower esophagus, the five-year survival is actually 34 per cent, and for those with a favorable growth in the midthoracic area (usually requiring a supra-aortic anastomosis), the survival is 14 per cent.

If the data regarding five-year survival can be considered to be comparable, which is questionable because of the small size of the roentgenotherapy series, it seems obvious that the weight of evidence is in favor of surgical removal of the disease.

It is doubtful, furthermore, whether the degree of relief from symptoms obtained by roentgenotherapy alone is as complete as that resulting from surgical extirpation. A comparison of the results of treatment by these two methods shows that with the former the relief of dysphagia is immediate and, in the majority of instances, permanent for as long as the patient lives, with only a few recurrences at the site of the anastomosis. With the latter, while a few have been able to take food of a liquid or soft-solid nature, the favorable result is usually obtained at the expense of considerable local pain or soreness during deglutition and often only with the aid of supplementary treatment by bougienage.

In conclusion, there is no evidence upon which to base a decision to abandon surgical extirpation in favor of roentgen therapy in the treatment of carcinoma of the esophagus. If the operative deaths are excluded and only those with an extent

of disease considered favorable for complete excision are included, the five-year survival following operation should be at least 34 per cent for patients with low esophageal lesions and 14 per cent for those whose growth is higher. The over-all five-year survival, if cases at all levels are included, should be 27 per cent. These results are not unlike those considered to be satisfactory in the treatment of carcinoma of certain other regions of the body.

It is apparent, therefore, that the current wave of discouragement regarding the surgical treatment of carcinoma of the esophagus is unwarranted.

RICHARD H. SWEET, M.D.

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SURGICAL REVERSAL OF SUPERIOR VENA CAVA SYNDROME

Report of Case Caused by Intrathoracic Goiter and Associated with Roentgenographic Hilar Vascular Shadow Simulating Neoplasm of Chest

B. A. McART, M.D.
F. B. RAMSEY, M.D.
W. A. TOSICK, M.D.
AND
K. R. WOOLLING, M.D., M.S. (Med.)
INDIANAPOLIS

SINCE William Hunter, in 1757, described a case of obstruction of the superior vena cava, many other cases have been reported in the world literature. Although the syndrome produced by such obstruction is relatively constant, the obstruction itself may result from many different primary diseases. Ehrlich, Ballon, and Graham [1] reviewed the world literature to 1933 on this subject and reported 309 cases. In their series, the etiologic agent in 35% of cases was aortic aneurysm, with or without arteriovenous fistula; in 50%, thoracic neoplasms, primary and secondary, and in 15%, rarer causes. In 1949, McIntire and Sykes [2] reviewed 219 cases and found the etiologic factor to be aortic aneurysm in 26.9%, primary and secondary thoracic neoplasms in 33%, chronic mediastinitis in 20.7%, and miscellaneous factors in 19.4%. Glushien and Mansuy [3] as well as Rose [4] have stressed the poor prognosis in vena caval obstructions, and the former authors reported that 70 to 85% were due either to malignant thoracic neoplasms or to aortic aneurysms. Benign lesions causing superior vena caval obstruction are relatively rare. The commonest type of benign lesion appears to be that of mediastinitis, with involvement of the superior vena cava either by cicatricial constriction or by pressure from inflammatory masses or from calcified hilar lymph nodes. Benign tumors are distinctly rare. Of 145 cases in which the primary etiologic agent was definitely verified, in the series of McIntire and Sykes, only 2 were found to be caused by benign tumors, 1 being a dermoid cyst of the mediastinum. Indirect trauma, such as that produced by heavy lifting,[3] presumably may initiate a chain of events in the superior vena cava analogous to that which occurs in the axillary vein as the so-called *thrombophlebité axillaire par effort*.

A brief consideration of the anatomical relations of the superior vena cava is helpful in understanding the pathogenesis of the syndrome. The superior vena cava is 7 cm. in length and extends from the junction of the right and left innominate veins to the right atrium. The terminal 2 cm. of the superior vena cava are intra-

From the Departments of Surgery (Drs. McArt and Ramsey), Radiology (Dr. Tosick), and Medicine (Dr. Woolling), of the Indianapolis General Hospital.

pericardial. The azygos vein poins the superior vena cava just before it enters the pericardium. In the anterior mediastinum, there is a very intimate relationship between the superior vena cava, esophagus, trachea, lymph nodes (bronchial, right anterior mediastinal, and right lateral tracheal), and aorta. Thus, any inflammation or tumor in these areas may cause direct focal pressure, constriction, invasion, or erosion of the superior vena cava. There may be complete obliteration of the lumen of the vessel by such pathologic processes, or the lumen may be narrowed yet maintain a degree of patency for a variable period of time. Thrombosis may be the principal cause of luminal closure, as following intimal damage from trauma, or it may only carry to completion a previously existing partial occlusion of nonthrombotic origin. If the resultant obstruction is above the entrance of the azygos vein, the subsequent collateral circulation may drain largely through the azygos vein into the right atrium. If, however, the obstruction is at or below the entrance of the azygos, the collateral circulation will drain ultimately into the right atrium via the inferior vena cava. The superior vena cava syndrome may develop as a result of bilateral occlusion of the innominate veins as well as occlusion of the superior vena cava per se.

The symptoms of superior vena caval obstruction are due fundamentally to venous hypertension in the region normally drained by this vessel. The symptoms vary in severity, depending upon the degree and location of obstruction and upon the extent of development of collaterals. The most prominent signs are the increased venous pattern and the cyanosis in the upper half of the body. The veins in the upper extremities, head, neck, and chest are dilated and prominent and exhibit increased venous pressure, as contrasted with the normal pressure in the veins of the lower extremities. If the obstruction is below the point of entry of the azygos vein, veins in the region of the abdomen are dilated as well. The direction of blood flow in the dilated veins in the chest and abdomen is downward. A tourniquet test has been described as clinically helpful in determining the location of the obstruction in relationship to the azygos vein.[5] A prompt and marked rise in the venous pressure in the arm after compression of the superficial collateral veins by a tourniquet placed tightly around the lower portion of the chest is interpreted as indicating that the obstruction is below the level of entry of the azygos vein. Varying degrees of cyanosis of the head, neck, trunk, and upper extremities are seen. The conjunctivae frequently are markedly injected. The term "Stokes' collar" has been applied to cyanosis, which is apparent mainly above the clavicles. The cyanosis presumably is due to increased prominence of the cutaneous veins and deoxygenation of the capillary blood due to venous stasis. In acute occlusions, marked edema of the face and neck may occur, but edema is not as striking in the more chronic occlusions in which there has been time for development of extensive collateral circulation. All symptoms are intensified with recumbency. Frequently, minimal cyanosis may be converted to severe cyanosis by instructing the patient to bend forward for several minutes. Many patients have dyspnea, orthopnea, and periodic bouts of hyperventilation which are thought by Altschule and co-workers [6] to be due to increased intracranial pressure with "tissue stasis of the brain." Such increased intracranial pressure is stated to be capable of producing severe headache, vertigo, tinnitus, somnolence, stupor, and Jacksonian or generalized convulsions.* Hemoptysis is an

* References 7 and 8.

attending symptom which, according to Friedberg,[9] is due to rupture of the tortuous and greatly dilated bronchial veins. Hussey and co-workers,[8] in a study of 34 cases of superior vena caval obstruction, reported venous pressure of 400 mm. in 12 cases, of greater than 300 mm. in 20 cases, and of less than 300 mm. in 2 cases. It is the consensus that the circulation time is a rather unreliable test for the diagnosis of this condition. Phlebograms may be of value in demonstrating the location of the obstruction and showing the extent of the development of collateral circulation † but are usually unnecessary in establishing a diagnosis of the syndrome.

REPORT OF A CASE

A well-developed, slender Negro man, aged 50 years, was first seen in the Peripheral Vascular Diseases Clinic, at Indianapolis General Hospital, on Dec. 16, 1952. He complained of swelling in the right leg, which had been present episodically for 10 to 12 years. Varicose veins had been present in both legs since youth. There was no definite history of thrombophlebitis.

Physical examination of the lower extremities disclosed bilaterally incompetent greater saphenous veins, moderate edema of the right leg, and slight edema of the left leg. The edema in the region of the right ankle was of the pitting type, but the skin over the shin region, which was indurated, scaly, warm, dusky-red-colored, and slightly tender, pitted only slightly. Stasis pigmentation was marked in the right leg and slight in the left, and there was a shallow ulcer measuring 1 cm. in diameter on the lateral surface of the right lower leg. There was no evidence of arterial insufficiency. The diagnoses were bilateral incompetence of the greater saphenous veins, bilateral chronic venous insufficiency and chronic indurated cellulitis and stasis ulcer of the right leg. The patient was admitted to the hospital for treatment of the varicosities and cellulitis.

Complete physical examination in the hospital disclosed, in addition to the findings noted above, large distended veins of the neck and upper extremities and a marked increase in the venous pattern of the chest (Fig. 1*A* and *B*). The direction of blood flow in the veins of the chest was downward. The conjunctivae were markedly injected, and the periorbital tissue was somewhat puffy. There was slight suffusion of the skin of the face and neck but no definite cyanosis. The thyroid gland was easily palpable, moderately firm, and multinodular, but it was not possible to delineate the lower poles of the gland. When the patient swallowed, both of the enlarged lobes of the thyroid could be felt extending downward to a substernal position. On recumbency, the increased venous pattern of the upper half of the body became more prominent, while in this position there was no evidence of increased venous pressure in the lower extremities. The results of the remainder of the physical examination were normal or negative.

On further questioning, the patient stated that he had noted fullness of the neck for the past 5 to 10 years and prominent veins of the neck and chest for a somewhat shorter period. He stated that his eyes had been "bloodshot" for several years and that during this time there had been gradually increasing tinnitus, impairment of hearing, hoarseness, and a sensation of "fullness in the head."

The results of routine enumeration of erythrocytes and leucocytes, estimation of hemoglobin, complete urinalysis, and serologic tests for syphilis were normal or negative. The total plasma cholesterol was 258 mg. per 100 cc. On Dec. 24, roentgenologic and fluoroscopic examination of the chest disclosed an increase in the size of the left hilar shadow, suggestive of bronchogenic carcinoma. There also was a homogeneous density in the mediastinum in the supracardiac region, with slight narrowing of the tracheal air shadow in this region, suggesting intrathoracic goiter (Fig. 2*A* and *B*). The basal metabolic rate on two occasions was +8. The venous pressure in a right antecubital vein was 190 mm. of saline. The arm-to-lung circulation time was 17 seconds, the arm-to-tongue time 22 seconds. An electrocardiogram was normal. Laryngoscopic examination was negative.

On Jan. 23, 1953, with the use of general endotracheal anesthesia, the region of the thyroid gland was explored, and a large intrathoracic goiter was found. Subtotal thyroidectomy was per-

† References 8, 9, 10, and 11.

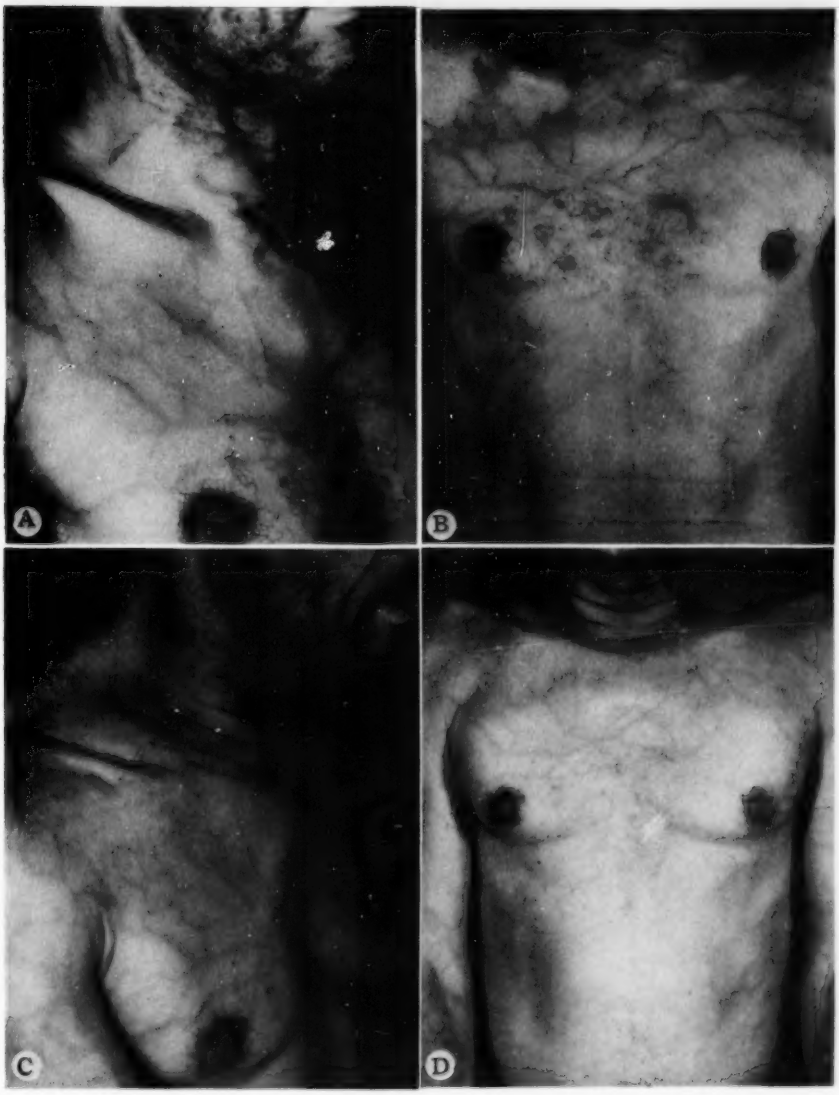

Fig. 1.—Superior vena cava syndrome. Infrared photographs showing venous pattern of increased prominence: *A* and *B*, oblique and anterior views, respectively, of neck and thorax before thyroidectomy; *C* and *D*, same views after thyroidectomy, showing decrease in prominence of veins.

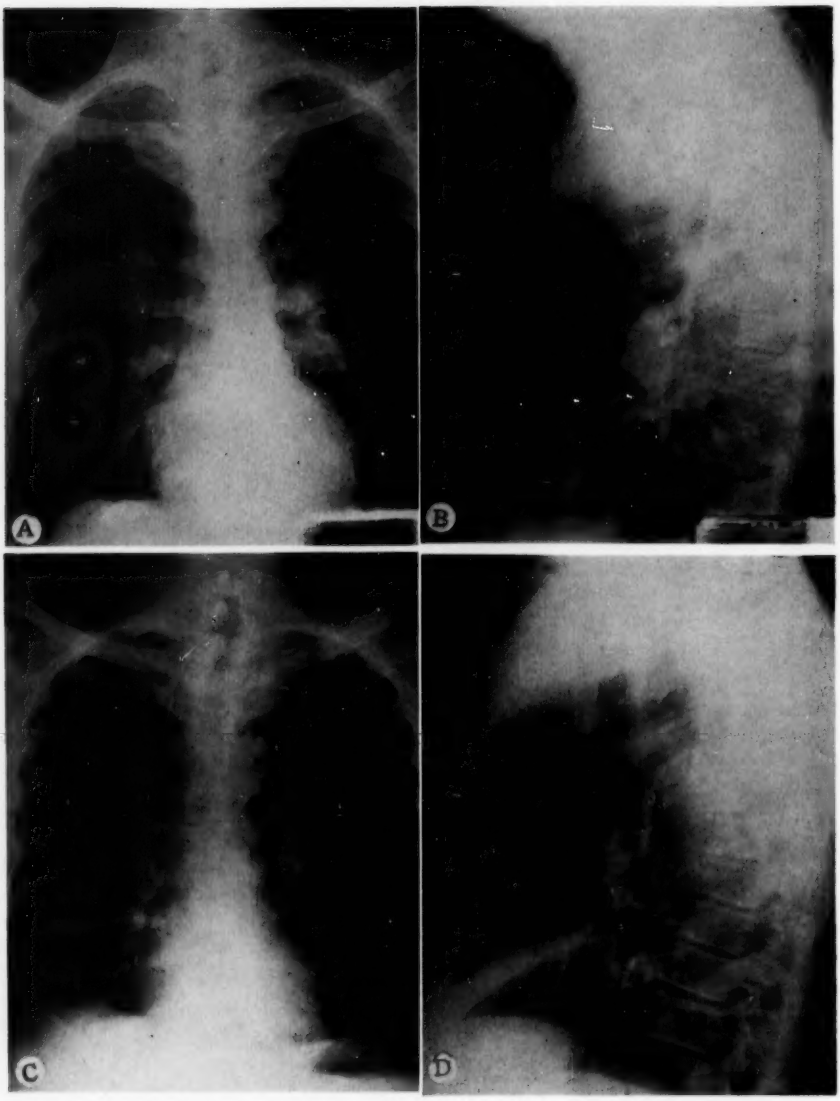

Fig. 2.—Superior vena cava syndrome. Roentgenograms of chest: *A*, posteroanterior view showing preoperative supracardiac mass with narrowing of tracheal air shadow and left hilar mass; *B*, lateral view showing preoperative left hilar mass; *C* and *D*, postoperative posteroanterior and lateral views, respectively, showing regression of left hilar mass.

formed, removing approximately seven-eighths of each lobe of the gland and the isthmus. The substernal portion on the right side was approximately twice the size of that on the left and measured 9 by 5 by 4 cm. (Fig. 3). The weight of the resected gland was 208 gm. Microscopic examination revealed a nodular colloid goiter of the "endemic" type (multiple adenomatous goiter).

Aside from evidence on the third postoperative day of partial atelectasis of the right lung, which subsequently resolved, the postoperative course was uneventful. On March 3, prior to dismissal, roentgenologic and fluoroscopic examination of the chest revealed a marked regression of the size of the left hilar shadow in comparison with the previous examination (Fig. 2*C* and *D*). The venous pressure in the right arm on Feb. 12, was 110 mm. saline. The marked engorgement and enlargement of the veins of the neck, upper extremities, and chest disappeared in the postoperative period (Fig. 1*C* and *D*). The cellulitis of the right leg was treated by elevation of the extremity and application of warm moist packs, with resultant healing. The preoperative sensation of "fullness" in the head, the tinnitus, impaired hearing, conjunctival injection, and hoarseness decreased considerably.

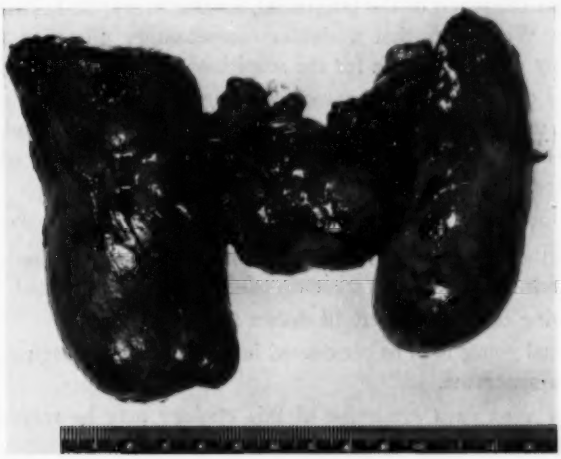

Fig. 3.—Photograph of surgically resected portion of intrathoracic adenomatous goiter which caused superior vena cava syndrome.

The patient was last seen in June, at which time he stated that he had gained 6 lb. (2.7 kg.) since release. Edema of the right leg had been well controlled by the wearing of a 75% rubber leg bandage. The patient was instructed to return in three months for consideration of surgical treatment of the incompetent greater saphenous veins and for reappraisal of the status of the thyroid.

COMMENT

In view of the clinical findings and the course, it is apparent that this patient had the superior vena cava syndrome. From the data available, one cannot be certain as to whether the obstruction was located in the superior vena cava proper or in both innominate veins. It is probable that the obstruction was above the level of entry of the azygos vein, since there were relatively few collateral veins in the region of the abdominal wall. The prompt and marked changes postoperatively indicate either that the obstruction had not been complete or, if complete, that complete occlusion by thrombosis had not occurred. The relatively low order of elevation of venous pressure would support the former view. The patient presented

a difficult problem in differential diagnosis. We were confronted with the superior vena cava syndrome associated with roentgenologic findings suggesting pulmonary neoplasm. The close causal relationship statistically of the latter to the former, with its attendant dire prognostic aspects, was well appreciated, yet the coincidental finding of a substernal goiter with serious tracheal compression offered a plausible alternative explanation for the vascular findings and a greatly brighter prognosis. The subsequent course almost certainly establishes that the alternative explanation was correct. We had noted in the literature only one previous reference to similar roentgenographic findings in the superior vena cava syndrome. This was the case reported by Friedberg,[9] in which a hilar mass was disclosed by roentgenographic examination of the chest. At autopsy, the patient was found to have had complete obstruction of the superior vena cava by calcified tuberculous mediastinal nodes, and the location of the roentgenographic shadow corresponded with the anatomic finding of great dilatation of the intercostal, accessory azygos, hemiazygos, and left phrenic veins. We believe that a similar compensatory dilatation of these veins almost certainly was responsible for the suspicious hilar shadow in our case.

As pointed out above, benign tumors causing the superior vena cava syndrome are relatively rare. We are not aware of a previous report of substernal goiter producing this syndrome, although it seems to us likely that this condition is not excessively rare.

SUMMARY AND CONCLUSIONS

A case of the superior vena cava syndrome, which was caused by substernal goiter and which regressed after resection of the goiter, is presented.

The following conclusions may be drawn from this study:

1. Substernal goiter is to be considered in the differential diagnosis of the superior vena cava syndrome.

2. Superior vena cava syndrome of this etiology may be reversed by proper surgical treatment.

3. Dilated hilar veins may be an integral component of the superior vena cava syndrome. Roentgenologically, these may produce a hilar shadow which may simulate that caused by pulmonary neoplasm. The shadow may regress after removal of the obstruction of the superior vena cava.

4. It is apparent that the discovery of the superior vena cava syndrome does not necessarily indicate a bad prognosis. The prognosis in each case can be established only by a determination of the fundamental nature of the obstructive lesion.

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SUCCESSFUL TREATMENT OF CARDIAC ARREST

Mitral Valvulotomy as the First Step in Resuscitation of the Heart

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THE INCREASING awareness that cardiac standstill is a reversible event has led to earlier recognition and successful restoration of the heart beat. Particularly in the realm of cardiac surgery, this heretofore catastrophic complication may be anticipated and efficiently treated. Except perhaps for such calamities as inadvertent occlusion of a coronary artery or sudden massive blood loss, there is usually ample advance evidence of an impending crisis presented to the alert anesthesiologist or cardiologist. An oscilloscopic device for continuous registration of the electrical activity of the heart under the constant observation of a competent interpreter is extremely desirable in cardiac surgery and in our experience has proved a significant factor in reduction of mortality.

Cardiac resuscitation may not be successful in the presence of advanced myocardial degeneration or inadequate coronary blood flow. However, in relatively normal hearts with adequate coronary arteries, resuscitation should be ultimately successful if adequate measures are immediately instituted. The restoration of the oxygen system depends upon the introduction of oxygen into the lungs and its circulation to the brain and other vital organs by effective massaging of the heart.[1] Thus, the system requires not only an adequate airway but also an adequate "blood-way." The presence within the circulatory system of mitral or pulmonic stenosis would of necessity retard efforts for resuscitation. The following cases illustrate the necessity for restoring an adequate "blood-way" in successful cardiac resuscitation.

REPORT OF CASES

CASE 1.—M. M., a 34-year-old white woman, was admitted to Mount Sinai Hospital of Cleveland with a history of acute rheumatic fever at the age of 12. Two years prior to present admission, a cerebral embolus resulted in right hemiplegia and aphasia. Mitral stenosis and auricular fibrillation were present. One year prior to admission a saddle embolus at the bifurcation of the aorta occurred. Embolectomy was performed by Dr. Lawrence Atlas. Residua of these embolic episodes were still present: right-sided hemiparesis and contracture deformities of both legs.

The patient was greatly incapacitated by shortness of breath on slightest exertion. She had taken digitalis for two years. Physical examination revealed the typical findings of mitral stenosis. The blood pressure was 110/80 mm. Hg. Auricular fibrillation with a ventricular rate of 84 per minute was present. At the apex, there was a snapping first sound and a rumbling diastolic murmur. The pulmonic second sound was loud and snapping. There was no clinical

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evidence of other valvular involvement. The lungs were clear, and there was no hepatomegaly or edema. The electrocardiogram showed auricular fibrillation, digitalis effect, and early right ventricular hypertrophy. Cardiac fluoroscopy revealed a prominent pulmonary artery and a greatly enlarged left auricle with elevation of the left mainstem bronchus.

Operation for mitral stenosis was performed on May 27, 1953, by Dr. Claude S. Beck. Injections of meperidine (Demerol), 75 mg., and scopolamine, 0.4 mg., were given subcutaneously one hour prior to operation. The patient was connected to the cathode-ray oscilloscope (Cardioscope) for continuous electrocardiographic recording. After induction of anesthesia with thiopental (Pentothal), sodium dicamethonium bromide (Syncurine), and cyclopropane were administered, followed by ether. The blood pressure was 90/50 mm. Hg with a heart rate of 60 per minute. Atropine, 0.2 mg., was given intravenously. Orotracheal intubation was followed by a heart rate of 140 per minute with a blood pressure of 100/58 mm. Hg. Neostigmine, 0.5 mg., was given intravenously and repeated after five minutes, after which the heart rate was 100 per minute and the blood pressure 100/60 mm. Hg.

The chest was opened by incision between the left fourth and fifth ribs. The intratracheal tube was connected to the Rand-Wolfe respirator. The heart rate was 80 per minute and the blood pressure 85/50 mm. Hg. The heart was exposed, and two purse-string sutures were placed at the base of the auricular appendage. Pressure was applied externally to the carotid arteries simultaneously with application of a clamp to the base of the appendage. The pulse rate dropped to 40 per minute and the blood pressure to 60/? mm. Hg. Atropine, 0.2 mg., was given intravenously. The surgeon was informed of imminent danger. The operation was stopped for 10 minutes while the lungs were inflated and then the auricular appendage was opened (no thrombi were found). Meanwhile, the ventricular rate increased to 120 per minute, and the blood pressure to 85/30 mm. Hg. Neostigmine, 0.125 mg., and lanatoside C (Cedilanid), 0.4 mg., were given intravenously. The operation was stopped again as the ventricular rate continued to increase to 140 per minute and then 180 per minute (supraventricular tachycardia), and subsequently ventricular tachycardia with a gradual fall in blood pressure to unobtainable levels occurred. After five minutes of ventricular tachycardia, procaine amide (Pronestyl), 500 mg., was given intravenously very slowly. Cardiac standstill occurred. In a few seconds, the surgeon introduced a finger into the left auricle. The valve orifice was about 6 mm. in diameter. The valve was rapidly opened to 2.5 to 3.0 cm. in diameter. A ligature was placed around the base of the appendage. Manual massage of the heart was then instituted with effective arterial pulsations palpable in the brachial artery throughout the procedure. Epinephrine, 5 cc. of 1 : 10,000 solution, was injected into the right ventricle simultaneously with intravenous injection of 5 cc. of 10% solution of calcium gluconate. Ventricular fibrillation appeared. Cardiac massage was continued while the electrodes of the Rand-Wolfe defibrillator were readied and tested. Two electric shocks (100 volts, 1.5 amp.) were applied to the heart with the defibrillator, and cardiac standstill reappeared. Occasional bizarre ventricular beats appeared, the contractile wave starting at the apex. Circulation was assisted with hand massage. The beats were unifocal and then supraventricular with aberrant conduction and more frequent (120 per minute, blood pressure 80/? per minute). The time elapsed from cardiac standstill to this point was approximately 10 minutes. Eventually a few short periods of regular sinus rhythm appeared, then reverted into auricular fibrillation with irregular ventricular rhythm as preoperatively.

The auricular appendage was amputated and its stump was sutured; the chest was closed, the pericardium being left open, since attempts to close it made it too tense, and the patient was sent to the recovery room with a heart rate of about 100 per minute and a blood pressure of 90/55 mm. Hg, with frequent premature ventricular beats. The patient recovered from the anesthesia without any apparent neurologic residua.

In view of the periods of regular sinus rhythm during recovery, it was thought that conversion to a regular sinus rhythm should be attempted postoperatively. On the fifth postoperative day, following an hourly dose of 0.2 gm. of quinidine sulfate for two doses, 0.4 gm. was given every three hours for three doses. This produced severe nausea and vomiting, and the attempt at conversion was discontinued at this time. The patient made an uneventful recovery and was discharged greatly improved on the 10th postoperative day.

Case 2.—B. B., a 2½-year-old white girl, was operated upon by Dr. Claude S. Beck at Mount Sinai Hospital of Cleveland for isolated pulmonic stenosis. An attempt was made to

do a direct transventricular valvulotomy with the Potts-Smith retractile valvulotome after probing the orifice with a metallic urethral sound. In incising the stenotic area, the anterior wall of the pulmonary artery was cut. Six sutures were placed in the pulmonary artery, and whole blood was rapidly infused. The heart went into ventricular fibrillation when the last stitch was being applied. Manual massage was started immediately, but it was felt that the ventricles were not effectively emptying. A Kelly clamp was introduced through the ventricular incision into the pulmonary artery and then opened in order to tear or dilate the orifice. Manual massage was restarted. The ventricles spontaneously stopped fibrillating. Hand massage was continued for about one hour. The heart had good tone and an idioventricular pacemaker took over, the contractile wave going from the left to the right ventricle. After a while the cardiac contractions were stronger, and a systolic pressure of 100 mm. Hg was obtained, with a sinus tachycardia. Two more stitches were placed on the pulmonary artery, which still leaked a small amount, and the chest was closed.

After one hour of observation in the operating room, the heart fibrillated again, the blood pressure falling to zero. The chest was opened again and heart massage was instituted. Electric shocks were given and the heart was defibrillated, but it was not possible to obtain a spontaneous heart beat. Another hour was spent attempting to restore the coordinated beat. It was believed that congenital deformities in the heart made successful resuscitation impossible.

On postmortem examination, the pulmonary valve orifice was found to be only a few millimeters in diameter with hypoplasia of the first 2 cm. of the pulmonary artery. The hemostatic sutures in the wall of the pulmonary artery further narrowed the outflow tract. A high ventricular septal defect was also present. The condition was inoperable.

COMMENT

The above two cases serve to illustrate the importance of attaining an adequate "blood-way" as an initial step in resuscitation of the heart. In Case 1, mitral commissurotomy was performed within seconds of the onset of cardiac standstill, and manual massage was instituted after the obstruction to left ventricular filling had been reduced. In Case 2, the obstruction to pulmonary flow was not relieved, and adequate right ventricular emptying, therefore, was not attained.

In attempting to resuscitate the heart, primary consideration must be given to restoring a normal blood volume and perfusion system. In those cases of cardiac arrest associated with severe blood loss, massage can be effective only if there is adequate venous return. This can be accomplished only by rapid reinfusion of blood (and secondary measures such as Trendelenburg position). Selection of the route of reinfusion depends upon only one consideration—that of the most rapid restoration of venous return. Consequently, it would appear that intra-arterial or intracardiac transfusion is unnecessary and inadvisable. Successful resuscitation depends not on the route of transfusion but on the adequacy.*

During cardiac surgery, arrest may occur as a result of traction or rotation of the heart so as to occlude large vessels.[4] Obviously, the restitution of normal anatomical position is the first step in restoring the heart beat in such a situation. In arrest associated with cardiac tamponade, intrapericardial pressure must be reduced to allow adequate venous return as the first step in resuscitation.

The physiological derangements associated with mitral stenosis originate from the physical obstruction of flow from left auricle to left ventricle with impaired diastolic filling of the ventricle. Obviously this inadequacy becomes a severe impediment to cardiac resuscitation in the presence of mitral stenosis. In Case 1, therefore,

* References 2 and 3.

it was felt that provision for adequate filling of the left ventricle could be made only by operative opening of the valve. Thus mitral valvulotomy was the first step in cardiac resuscitation.

The high incidence of cardiac fatalities in surgery for pulmonic stenosis (Cooley reports 48 deaths in 878 cases) [5] is further evidence of the difficulty in reestablishing the heart beat in the presence of significant obstruction to blood flow.

As Beck has repeatedly pointed out, restoration of the heart beat is neither difficult nor urgent, once the oxygen system is restored,[1] accomplished by delivering oxygen into the lungs and then circulating the oxygen by squeezing the heart. In the successful case herein presented, standstill occurred at the propitious moment, namely, after all preparations had been made for entering the left auricle. The chain of events leading up to standstill had been recognized and anticipated.

Ventricular fibrillation is a frequent occurrence either at the onset or during resuscitation in cardiac arrest during cardiac surgery. (It probably occurs more frequently than is recognized.[6]) The onset of ventricular fibrillation is most readily recognized electrocardiographically even before the surgeon is aware of its occurrence. In the case presented, the immediate recognition and treatment of fibrillation by counter shock was a factor in the successful outcome.[7]

The simultaneous application of pressure to both carotid arteries is a routine procedure in attempting to prevent embolization to the brain during manipulation of the atrium. In Case 1, the chain of events leading to standstill appeared to follow this maneuver, perhaps indicating the inherent danger of bilateral carotid sinus stimulation.

A few words might be said about the use of certain cardiotropic drugs in regard to cardiovascular surgery and cardiac arrest. The administration of procaine amide was undertaken as an emergency procedure in Case 1. We do not favor its routine use. The occurrence of arrest after 500 mg. was given may have been precipitated by this drug. However, the subsequent ease of defibrillation and even the temporary period of sinus rhythm during recovery were probably beneficial effects.

The use of neostigmine and/or lanatoside C (even after complete digitalization) is justified in supraventricular tachycardia when the ventricular rate becomes too rapid to allow adequate diastolic filling in mitral stenosis. In our opinion, routine digitalization should be carried out in all patients with mitral stenosis coming to surgery. In our experience, digitalis does not increase the irritability of the heart during surgery. As a matter of fact, routine digitalization of patients with coronary disease (without failure) undergoing the Beck coronary operation has reduced to zero the mortality during operation. Ectopic rhythms have been significantly diminished since digitalis has been used routinely. As a result of these observations, we have undertaken a program of routine digitalization of all patients over the age of 50 years undergoing major surgery at Mount Sinai Hospital of Cleveland. A preliminary survey indicates that this measure has reduced the cardiovascular complications in these patients.

Atropine sulfate in intravenous doses as small as 0.1 mg. and as large as 3 mg. has proved extremely effective in preventing vagovagal episodes. The appearance of a slow nodal rhythm or even a sinus bradycardia (below 70) should be interpreted as a danger sign, and atropine should be given. A salutary effect can be

observed within a few seconds after intravenous administration. We recommend the use of atropine before intubation of the trachea is carried out and before the patient is placed in position for operation.

Calcium gluconate in doses of 5 to 10 cc. of 10% solution intravenously, presumably as a result of the change in the Ca^{++}/K^{+} ratio, repeatedly has increased the "tone" of the myocardium and prevented the heart from becoming flabby, at the same time increasing its inherent rhythmicity.

Although epinephrine aids in restoring a strong and frequent ventricular beat, it greatly increases the tendency to ventricular fibrillation.[7] Certainly the anoxic heart is prone to epinephrine-induced fibrillation. In our early studies with mephentermine [8] (Wyamine), it was found that even extremely large doses of this pressor amine failed to produce increased ventricular irritability in dogs and humans. Large doses in patients with shock accompanying myocardial infarction produced a pressor response but no increase in ventricular irritability.[9] In patients undergoing cardiovascular surgery, the routine use of mephentermine has resulted in gentle vasopressor effect.[8] Furthermore, in cases of cardiac arrest in which mephentermine had been used, it was noted that large, frequent doses of epinephrine were not followed by ventricular fibrillation. Recent studies by Dr. Joseph Seifter and his co-workers † have demonstrated the effectiveness of mephentermine in preventing epinephrine-induced arrhythmias in the isolated frog heart and in the intact dog. The possible therapeutic usefulness of these observations is being investigated.

Despite increased awareness, cardiac arrest has become a greater diagnostic and therapeutic problem.‡ Cardiovascular surgery obviously carries the greatest danger of cardiac arrest. Proper selection and preparation of patients for heart surgery and supervision during operation can prevent this catastrophe, or at least render it amenable to treatment. Continuous electrocardiographic observation is highly desirable, as is oxymetric determination of changes in arterial oxygen saturation.

Training in resuscitation of the heart appears to be a prerequisite to the performance of surgery.[14]

SUMMARY AND CONCLUSIONS

Successful cardiac resuscitation was performed in a patient undergoing mitral valvulotomy. Cardiac standstill was anticipated, and mitral valvulotomy was successfully performed immediately after cessation of the heart beat, thus establishing an adequate "blood-way" for resuscitation. In another patient, restoration of the coordinated heart beat was temporarily successful. Abnormalities in the heart were hypoplasia of the pulmonary artery and a ventricular septal defect. These abnormalities precluded success. The attempt to restore the coordinated beat should not be stopped until every effort has been made. In this case, the chest was reopened and several hours were spent in the resuscitation procedure.

Successful resuscitation depends upon restoration of the oxygen system before irreversible damage occurs in the brain (three to five minutes). Restoration of the oxygen system requires delivery of oxygen into the lungs and circulation of oxygen by squeezing the heart. This is an emergency procedure. Restoration of the heart

† Personal communication to the authors.

‡ References 6, 10, 11, 12, and 13.

beat follows. This part of the procedure is not an emergency as long as the oxygen system is maintained. The continuous electrocardiogram was helpful in these cases, and its use is recommended.

Restoration of the heart beat is aided by the judicious use of such cardiotropic drugs as epinephrine, procaine amide, atropine, calcium gluconate, neostigmine, and lanatoside C.

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ACUTE NECROTIZING GASTRITIS

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OF THE SEVERAL types of acute gastritis, the rarest is acute necrotizing gastritis. The case to be reported is of unusual interest because it appeared to originate in an overwhelming periodontal infection, complicated by ulcerative stomatitis, with a necrotizing infection spreading to the esophagus and stomach, despite the fact that the major arterial vessels supplying them were not occluded.

REPORT OF CASE

The patient was a well-developed man, aged 63. He was admitted to the Albert Einstein Medical Center, Northern Division, on Sept. 26, 1952. Chief complaint on admission was vomiting of large quantities of blood-stained fluid, and "pain in the stomach and over the throat."

History of Illness.—On the day prior to admission the patient went to work as usual. He ate dinner with his family that evening. There was nothing unusual about the meal except that he ate some mushrooms which were picked by a neighbor. However, he ate very few of these. The other members of his family and those of the donor's family ate considerably more of the mushrooms than did the patient, and there was not a single instance of unusual symptoms in any of these persons. After dinner the patient had a couple of rye highballs, which was his usual custom. In this he was joined by other members of the family. He went to bed feeling well, but was awakened during the night by nausea, and he vomited but felt too weak to get out of bed. Consequently, when his family came to wake him in the morning, they found a great deal of blood-stained vomitus in the bed. The patient complained of extreme weakness at this time. His physician was notified and promptly sent him to the hospital.

Past Medical History.—The patient is known to have had lead poisoning at the age of 27. In 1928 he was in an automobile accident and suffered a cerebral concussion, from which he recovered. During the past year he had lost 30 to 35 lb. (13 to 15 kg.) in weight. His appetite had been poor for the past five weeks. He complained of frequent pains in the abdomen, which were not relieved by food. However, he had not seen a physician for the past 20 years. His home life was quite happy, and he denied that he had taken any unusual medicine or that there was any attempt at suicide. This statement was corroborated by his family, who stated that there were no toxic drugs or poisons available to the patient in the house.

Social History.—The patient had been a painter for many years. Lately he had been employed spraying paint (nontoxic) on automobiles and had missed no days at work. He had three children living and well. He had been accustomed to having a couple of drinks of whiskey or beer each day. He had been an inveterate pipe smoker. "He always had a pipe in his mouth."

Family History.—One sister died of cancer. Two sisters are living and well. One brother is living and well. There is no history of tuberculosis or allergies. His father died of pulmonary embolism and his mother died at childbirth.

Attending Surgeon (Dr. Behrend), Associate in Medicine (Dr. Katz), and Assistant Pathologist (Dr. Robertson), Albert Einstein Medical Center, Northern Division.

Fig. 1.—Photograph shows severe periodontal infection and ulcerative stomatitis (first postoperative day).

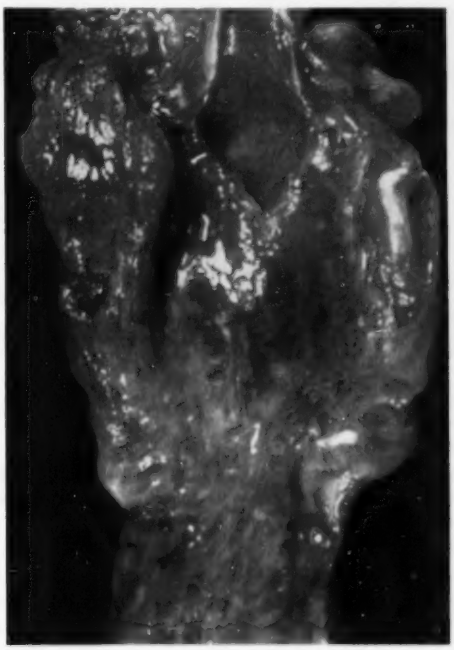

Fig. 2.—Postmortem photograph showing necrosis and ulceration at proximal esophagus.

Physical Examination.—On admission, the blood pressure was 130/80, pulse 72, respiration 25. The patient was lying quietly in bed and obviously was acutely ill. He appeared to be only semiconscious. It was noted that the lips were caked with dry blood. The lungs and heart were normal. The abdomen was moderately distended, especially in the upper portion. There was generalized pain and tenderness throughout the abdomen, most marked, however, in the epigastrium. Peristalsis was absent throughout. White blood cell count on admission was 12,300, with 86% polymorphonuclears. The red cell count was 4,470,000 (hemoglobin 13.4 gm.). Serum amylase was 333 units. The flat plate x-ray of the abdomen showed only gastric dilatation. No air was observed under the diaphragm. A Levin tube was passed into the stomach, and a considerable amount of dark-brown blood-stained fluid was aspirated, which was alkaline in reaction. A diagnosis of peritonitis of unknown origin was made, and operation was advised.

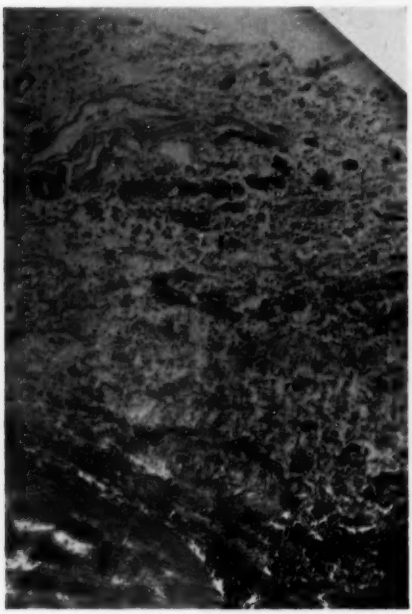

Fig. 3.—Photomicrograph of stomach showing destruction of mucosa and necrosis of all layers.

Fig. 4.—Another microscopic section of the stomach showing necrosis and infiltration of all layers by leucocytes.

Operation.—With the patient under intratracheal anesthesia, a right upper pararectus incision was made. Approximately 2,000 cc. of muddy fluid was removed from the peritoneal cavity. It was similar to the fluid aspirated from the stomach. A specimen was obtained for culture. After removal of the fluid the stomach became visible. It was of uniformly gray-green color, and the color was similar to that of intestine normally seen in an autopsy specimen. The entire stomach, on both anterior and posterior walls, appeared to be nonviable. The lesser peritoneal cavity was exposed and found to contain a considerable amount of fluid. Some infected fluid was also aspirated from above the liver, underneath the liver, from between the loops of intestine and from the pelvic cul-de-sacs. There was a sharp line of demarcation 0.5 cm. proximal to the pyloric vein. Beyond this the duodenum appeared normal. The liver was of normal color. An attempt was made to find a perforation of the stomach, and the contents of the stomach were compressed manually, but it was impossible to force any of the gastric contents from within its lumen by this maneuver. It was felt that the fluid in the peritoneal cavity had arrived

there by transudation through the devitalized wall of the stomach. The stomach wall was of normal thickness; there was no phlegmon. The patient's condition on the operating table rapidly became so poor that no thought of total gastrectomy could be entertained. Jejunostomy was considered, but it was felt that the patient's course would be rapidly fatal and that he would not live to derive much benefit from this procedure. Therefore, two large tubes were inserted through stab wounds for drainage, one on each side of the abdomen. The abdomen was closed with through-and-through sutures. During the operation the patient received 500 cc. of whole citrated blood. A million units of penicillin and 1 cc. of a penicillin and dihydrostreptomycin preparation (Combiotic) were given twice daily. Because of the unusual nature of the case, a specimen of the gastric contents was aspirated postoperatively for analysis, but the examination was not made.

On the day following operation, the patient developed auricular fibrillation. On Sept 28, he felt considerably better. A dental consultant (Dr. A. R. Leinweber) was asked to see the patient. A diagnosis of gangrenous stomatitis was made. Pentabs, containing 200,000 units of penicillin were given every two hours, by mouth. The following day the patient was quite apathetic, although the condition of the mouth appeared to be improved. (ACTH) Corti-

Fig. 5.—Photomicrograph of esophagus, showing destruction of mucous membrane and necrosis of submucosa.

cotropin, which had been started immediately after the operation, was discontinued because of the danger of perforation of the stomach. The abdominal pain and tenderness had by this time disappeared, with the exception of some residual tenderness in the epigastric region. Despite repeated transfusions and supportive parenteral therapy, the patient's condition steadily deteriorated, and he died on Oct. 4 (eighth postoperative day).

Bacteriologic Examination.—At operation, smears and culture from the peritoneal cavity showed fusiforms and hemolytic and nonhemolytic staphylococci. Smears and culture made by the dental consultant showed Escherichia coli and Bacillus pyocyaneus and Fusobacteria, Nocardia and Gram-positive and negative cocci and bacilli in chains.

At autopsy, smears from the gastric mucosa were taken and showed spirochetes and Fusobacteria. Homogenates from the stomach also showed Fusobacteria and Gram-negative rods. Cultures showed B. pyocyaneus, Aerobacter aerogens, and Proteus morganii. A smear from the lung showed Fusobacteria, and on culture B. pyocyaneus was grown. There was no growth from the heart's blood culture. In short, the same types of organisms were cultured from the mouth, base of the teeth, gastric mucosa, and the peritoneal fluid.

Autopsy Examination.—Permission for autopsy was obtained, and the main findings were ulcerative stomatitis and glossitis, a necrotizing esophagitis with secondary mediastinitis, and

complete necrotizing gastritis involing the entire stomach. There were evidences of ulceration only in the mouth and proximal 3 in. (7.6 cm.) of the esophagus. In the remainder of the esophagus and stomach the process was one of diffuse and total destruction of the mucous membrane in both organs. There was no occlusion of the arterial circulation of the stomach. Thrombosis of the portal system was found. This was felt to be an antemortem rather than a postmortem finding, since the clot was well organized.

COMMENT

Acute gastritis may be classified as follows: (1) simple (due to alcohol, drugs, allergens); (2) corrosive (due to strong caustics, acids, other chemicals); (3) infectious (associated with influenza, pneumonia, scarlet fever, etc.); (4) phlegmonous (due to suppurative infection by Streptococcus, colon bacillus), and (5) necrotizing (due to infection by necrotizing bacteria such as fusiforms and spirochetes).

The necrotizing form of gastritis may also be seen in conjunction with gastritis of a corrosive nature due to the swallowing of caustics and acids. It appears to be caused by bacterial infection alone very uncommonly. Many leading textbooks of pathology fail to mention necrotizing gastritis, and in a few instances in which mention is made of it, it is noted simply as being a very uncommon and practically always fatal condition.

The outstanding predisposing factor to infection in this case appears to have been very poor oral hygiene. While the symptoms that brought him to the hospital were fulminating, there was a history of 30 to 35 lb. weight loss and frequent abdominal pain in the past year.

One intriguing point in the history is the fact that he ate mushrooms gathered by a neighbor just prior to the onset of the acute symptoms. We are inclined to consider this of no contributory significance since others ate the same mushrooms, and indeed, more of them than the patient. However, Reese [1] notes that "the very same fungi have acted on some members of the family as vomitants only, on others as narcotics. Orfila relates a case of poisoning of a family of six persons by Amanita citrina. Wife, servant and one child had vomiting, followed by deep stupor, but they recovered. The husband had violent cholera, but he recovered also. The two other children became profoundly lethargic and comatose, and death ensued."

The literature contains many reports of phlegmonous gastritis.* These are usually characterized by localized areas, single or multiple, of thickening of the stomach wall with abscess formation between the layers, involving mainly the submucosa. The stomach wall shows evidence of diffuse inflammation. In contrast, the case here reported showed a pale, atonic, and necrotic gastric wall.

Rosenthal and Tobias [9] describe a case in some aspects similar to ours. This was an 18-year-old youth with pseudohypertrophic muscular dystrophy, who was admitted to the hospital because of abdominal pain, which disappeared, but returned four days later, accompanied by coffee-ground vomitus. Operation showed huge gastric distention. There were two large areas (6 cm.) of frank necrosis through which blood and gastric contents escaped. Four or five smaller areas of necrosis were noted. Liver and gall bladder were not involved. At autopsy the esophagus was normal. The authors noted a violent necrobiotic process similar to that of an infarction, but careful search failed to disclose thrombosis or occlusion of any

* References 2 to 8.

vessel. In this latter respect our case is comparable. There was no evidence of interference with the rich arterial blood supply of the stomach, yet the necrotizing infection was so virulent and diffuse that the integrity of the gastric wall was sufficiently compromised to permit the escape of gastric contents without any evidence, even at autopsy, of gross perforation. Another interesting fact noted was the sharp line of demarcation between necrotic and healthy tissue at the pylorus. This we were unable to explain. It does not seem likely that the difference between the normal pH of stomach and duodenum could alone limit the extension of an infection which involved all layers of the stomach.

Smith [10] found no evidence that the spirochetes of the mouth can, by themselves, produce disease in man. He considered them the most important members in a symbiotic group of anaerobic organisms capable of initiating a severe and fatal disease. They may produce such diseases as noma, fusospirochetal pulmonary gangrene and abscess, tropical ulcer, and pseudomembranous and phagedenic infections of the male and female genitalia. Smith also noted that they may involve eyes, ears, trachea, bronchi, esophagus, stomach, and intestines, and when carried in emboli in the blood stream, they initiate disease in the brain and other organs.

A few cases of spirochetal disease of the gastrointestinal tract have been reported.† In most instances these were associated with severe fusospirochetal infections of the mouth, pharynx, and lungs, or with cancer of the esophagus or stomach. Broughton-Alcock's [13] case began with abdominal pain, vomiting, and bloody diarrhea. There was marked prostration, and the patient died about a week after the onset of symptoms. At necropsy, localized abscesses were found between coils of the ileum and the ascending colon, and parts of the bowel appeared gangrenous. On the necrotic surface, cocci and a few spirochetes were found. There was very little cellular reaction to the infection. In some places a few polymorphonuclears were noted, in others only round and epithelioid cells.

The association of fusospirochetal infection with ulcerating cancers of the intestinal tract has also been noted by Wolkoff and Zinserling,[14] Lauda,[15] and Luger and Neuberger.[16]

SUMMARY

A unique case of acute ulcerative stomatitis complicated by necrotizing esophagitis and gastritis has been presented. Review of the literature emphasizes the rarity of this type of infection. Despite large doses of antibiotics, death resulted. Cultures from the mouth, esophagus, stomach, and peritoneal fluid revealed fusiforms, spirochetes, and cocci in all cultures.

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BASAL CELL CARCINOMA OF THE ANUS

Report of a Case

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ST. LOUIS

BASAL cell carcinoma of the anus is a lesion that has been diagnosed and reported in the literature only 12 times. Basal cell carcinomas are classically described as occurring on the skin of the face and head superior to a line drawn posteriorly from the angles of the mouth. Experience teaches us that basal cell carcinoma may occur anywhere on the body, on the covered as well as on the exposed surfaces. Its occurrence in the anal area is most rare, and so a 13th case is published here. The previously reported cases are summarized in the accompanying Table.

Results of Various Treatments of Basal Cell Carcinoma of Anus

Author	Age	Sex	Lesion	Treatment	Result
Buie and Brust, 1933.....	53	F	Orange-size lesion with inguinal mass	Refused	Unknown
	53	F	Advanced, annular	Colostomy with posterior resection	Died; recurrence, 9 mo.
Guess, 1935	52	M	2 cm., circumscribed	Local incision and radon seeds	Died of coronary occlusion, 5 mo.; no recurrence
Keyes, 1937	52	F	Local mass	Excision and x-ray	Died of recurrence
Tucker and Hellwig, 1938.	43	M	2 cm., circumscribed	Local excision and radium	No recurrence, 3 yr. postop.
Lawrence, 1941	73	M	Annular, invading prostate and urethra	Palliative colostomy and posterior resection	Died; recurrence, 2 yr.
Lawrence, 1941	73	M	Annular, invading perirectal spaces	Colostomy and posterior resection	Died; postop.; no autopsy
Gabriel, 1946	70	M	1 to 2 cm., circumscribed	Local excision	No recurrence at 1 yr.
Bacon, 1949	60	M	Local mass	Abdominoperineal resection	Died; recurrence, 19 mo.
	52	F	Local mass	Resection and node dissection	Died, 9 mo.
Lott and Alexander, 1949.	62	F	Induration of mucosa 1 × 1 cm.	Excision and 5,500 r x-ray	No recurrence 6 mo. follow-up
Hall, 1952	58	M	Polypoid, 1.5 cm.	Excision	No recurrence to date

From the Surgical Service of the Barnard Free Skin and Cancer Hospital.

REPORT OF CASE

A 65-year-old white man (Case No. A 14755) was first seen at the Barnard Free Skin and Cancer Hospital on July 25, 1952, with the chief complaint of a "sore on his rectum." Three years prior to his admission to the outpatient department of this hospital, he had a fistula in ano which was treated by excision. The excision specimen was reported pathologically as basal cell carcinoma, but apparently was not followed up. The wound healed, and the patient had no complaints until about July, 1951, when the soreness recurred in the operative site. His history was otherwise noncontributory.

The only significant physical finding was the granulomatous tissue with central ulceration extending around the posterior anal margin. The scar of the fistulectomy was apparent, and at

Photomicrograph of basal cell carcinoma of the anus.

its lateral pole was a nodule about 1 cm. in diameter. Biopsy of this nodule, as well as the friable granulomatous tissue was reported as basal cell carcinoma (Figure).

In view of the diagnosis, it was felt that this lesion should be radiosensitive, and radiation therapy was carried out. Because of the moist nature of the skin of this region, it was decided to treat the lesion slowly; however, by the time 2,500 r had been given to the area by the x-ray department, moist epithelitis had occurred. Therapy was stopped for several weeks until this subsided, and then 2,500 r was again given, making a total of 5,000 r. Biopsies at this time revealed the tumor to be largely unaffected. On Jan. 24, 1953, a one-stage abdominoperineal resection was done. The tumor was reported as locally invading the sphincters and had grown to the mucocutaneous junction. All lymph nodes were free of tumor tissue. The tumor was reported as adequately excised.

The postoperative course has been uneventful, aside from the protracted healing attendant upon the wide excision necessary. To date (January, 1954) there is no evidence of recurrence.

COMMENT

This case is reported, not only because of its rarity but also because of the failure of x-irradiation. As pointed out by Ackerman and Del Regato, basal cell carcinomas are radiosensitive. It was with this concept and in the hope of preserving the normal sphincter function that radiation therapy was carried out. So little radiation effect was noted on the subsequent biopsies, after 5,000 r, that it was deemed useless to continue the therapy, and resection was done.

There are not a sufficient number of reported cases of anal basal cell carcinoma to arrive at any definite conclusion as to the proper method of therapy. Obviously, the hope of cure depends upon the extent of the disease. The one-stage abdominoperineal resection has no advantage over the colostomy and posterior resection from a therapeutic standpoint, because one is not usually concerned about the regional lymph nodes. In our hands, the one-stage abdominoperineal resection has carried no greater mortality, and perhaps less morbidity, than the two-stage procedure. In conclusion, if properly applied irradiation is not successful, then some forms of radical resection is necessary.

SUMMARY

A case of basal cell carcinoma of the anus is reported. An attempt was made to cure the lesion with x-rays, but this failed, and abdominoperineal resection was done.

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ACUTE INTESTINAL OBSTRUCTION SECONDARY TO MECKEL'S DIVERTICULA

Report of Nine Cases

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JOHANN FRIEDRICH MECKEL, a German anatomist and surgeon, in a series of writings between 1808 and 1812 * gave the first adequate description and explanation of the embryologic origin of the diverticulum which now bears his name, but its existence has been previously referred to in the medical writings of Hildanus, Lavater, Ruysch, Littré and Morgagni.[11] Today it is known to be one of the commonest congenital anomalies, occurring in 1 to 3% of all cases in which necropsy is performed.

In the past 75 years Meckel's diverticulum has occupied a prominent place in the medical literature, not because of the anomaly itself, but because of the serious surgical complications which may arise from the diverticulum and manifest themselves in a number of ways. The most frequent clinical picture is that of massive intestinal hemorrhage, followed in turn by acute or chronic intestinal obstruction, localized or generalized peritonitis, vague and nagging abdominal pain without demonstrable pathologic features, mass in the abdomen, or umbilical fistula.

That intestinal obstruction could result from a Meckel diverticulum became well known toward the end of the 19th century, for in 1902 Halstead was able to collect from the literature data on 68 cases, to which he added 1 of his own, and in 1913 Wellington collected data on 212 cases. The manner in which the intestine may become obstructed by a Meckel diverticulum is of great importance to the surgeon, and a thorough knowledge of the possibilities in any individual case will often permit him to give prompt relief by directing him at once to the seat of the obstruction. In general, intestinal obstruction secondary to a Meckel diverticulum will fall into one of the following categories: (1) adhesive bands; (2) intussusception; (3) volvulus; (4) cicatricial narrowing secondary to ulceration or diverticulitis; (5) long diverticulum tied around the bowel as a noose or knot; (6) angulation of intestine at the point of attachment of a distended or cystic diverticulum; (7) prolapse of intestine through an umbilical fistula; (8) enterolith or gallstone extruded from the diverticulum into the intestine, and (9) contents of a hernial

The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

Assistants to the Surgical Staff (Drs. Chance and Hood), and members of the Section of Surgery (Dr. Waugh), Mayo Clinic and Mayo Foundation.

* References 12 to 14.

sac. To this list we would like to add another factor which was seen in three of our cases, namely an acute localized peritonitis secondary to peptic ulceration or diverticulitis with perforation.

In a five-month period three patients were seen on the emergency surgical service at the Mayo Clinic with a picture of acute intestinal obstruction, and at the time of operation complications of Meckel's diverticula were found. Reviewing the records of the clinic from 1940 to 1952, inclusive, we found nine patients who presented with an acute intestinal obstruction secondary to Meckel's diverticula, but in none was the mechanism of obstruction similar to the three cases referred to above. The manner in which the intestine became obstructed in these nine cases readily divides them into three groups. The first, consisting of three cases, includes those in which the obstruction was due to a localized inflammatory process secondary to peptic ulceration with perforation in two cases and diverticulitis with perforation in one case. The second group, consisting of four cases, includes those in which the diverticulum or its terminal ligament was attached to some viscus, thereby producing an internal opening through which prolapsed a loop of small bowel which became obstructed. In the third group were two cases in which the diverticulum was the leading point of an intussusception.

REPORT OF CASES

Group 1

Case 1.—A 43-year-old farmer was admitted to the emergency service on Nov. 30, 1952, with a three-week history of recurring bouts of cramping abdominal pain, distention, nausea, and vomiting. An appendectomy done elsewhere the day after initial onset of symptoms had not altered the course. For five days before admission the patient had vomited everything he took by mouth, became markedly distended and had passed nothing by rectum. There had been a loss of 20 lb. (about 9 kg.).

Physical examination on admission revealed a markedly dehydrated man who was acutely ill. The temperature was 99 F., pulse rate 84, and respiration and blood pressure normal. The abdomen was markedly distended and tympanitic, with high-pitched peristaltic sounds on auscultation. There was a moderate amount of tenderness with rebound in both the right and left lower quadrants, and on rectal examination there was tenderness, but no masses could be felt.

Laboratory studies showed the urine to be normal, hemoglobin 12.2 gm. per 100 cc. of blood, leucocyte count 3,900 per cubic millimeter of blood with a differential count that showed some increase in the band polymorphonuclear leucocytes. The sedimentation rate was 25 mm. in one hour, urea 22 mg. per 100 cc. of blood, chlorides 99.7 mEq. per liter, carbon dioxide 31 mEq. per liter, flocculation reaction for syphilis negative, and the patient's blood was Group A, Rh-positive. A roentgenogram of the abdomen was reported as showing dilated gas-filled loops of small bowel consistent with intestinal obstruction (Fig. 1).

In the hospital decompression was done with a Miller-Abbott tube, and the patient was given intravenous fluids and electrolytes. His general condition improved remarkably, and on the third day a proctoscopic examination disclosed an extrarectal mass at the 18 cm. level. On the following day a roentgenogram of the colon was negative, but the terminal ileum could be filled only 6 in. (about 15 cm.), consequently, an obstructing lesion there could not be ruled out. An attempt to clamp the long tube was made, but when cramping abdominal pain rapidly developed, exploration was decided on.

On Dec. 6, exploration was carried out by one of us (Hood) through a right lower rectus muscle-retracting incision. A large Meckel diverticulum, in which there was diverticulitis and a free perforation with a localized pelvic peritonitis, was found to be the site of the intestinal obstruction. There was a fibrous cord from the tip of the diverticulum to the umbilicus, but this was in no way related to the obstruction. The Meckel diverticulum, along with 15 cm. of ileum, was resected and an end-to-end ileoileostomy over a three-bladed clamp was carried out. The

pathologist reported that the Meckel diverticulum, which measured approximately 7 by 2 cm., showed acute diverticulitis and peridiverticulitis and that a large part of the wall above was destroyed by the inflammatory process. The remaining portions of the wall were densely adherent to the adjacent ileum.

Convalescence was somewhat prolonged because of the marked pelvic peritonitis, which responded to antibiotics and physical therapy. When dismissed on the 22d postoperative day the patient was afebrile, eating well, and having normal stools. When he was last heard from, his recovery was complete.

CASE 2.—A 5-month-old boy was first admitted to the pediatric service on June 30, 1952, because of a febrile illness of 24-hours' duration and the passage of a bloody stool. Except for a rectal temperature of 103 F., the results of physical examination were within normal limits.

Laboratory studies included a urinalysis, hemoglobin and erythrocyte count, all of which were normal. A roentgenogram of the chest was reported as negative.

The febrile reaction was thought to be on the basis of a respiratory infection which was treated with 300,000 units of procaine penicillin G (Duracillin) daily, and the patient rapidly

Fig. 1.—Dilated gas-filled loops of small bowel consistent with intestinal obstruction.

became afebrile. A proctoscopic examination was negative to 19 cm., and x-rays of the colon were reported within normal limits. It was felt that a Meckel diverticulum was the most likely diagnosis, but because of the recent febrile illness the patient was dismissed to return in three months for exploration.

On July 20, the infant was readmitted with a 48-hour history of cramping abdominal pains, abdominal distention, fever and vomiting. No gas or stool had been passed for the previous 24 hours. On physical examination the rectal temperature was 100.6 F., the abdomen was moderately distended and tympanitic with occasional high-pitched bowel sounds, and there was moderate generalized muscle guarding. On rectal examination there was the suggestion of a mass high on the right side. The hemoglobin content was 10.6 gm. per 100 cc., chlorides 93.2 mEq., and carbon dioxide 16 mEq. A roentgenogram of the abdomen revealed multiple gas-filled loops of bowel with fluid levels (Fig. 2).

The patient was prepared for operation by correction of fluid and electrolyte imbalance, gastric suction, and administration of antibiotics. The abdomen was explored by one of us (Chance) through a right upper quadrant transverse incision just above the umbilicus, and the patient was found to have a Meckel diverticulum with a perforation of a peptic ulcer at the base which had localized in an abscess surrounded by several loops of small intestine. The

abscess was evacuated, the diverticulum was excised, and the ileum was closed transversely. The pathologist reported that the Meckel diverticulum measured 2.5 by 1.5 by 1.5 cm. Its distal portion was lined by gastric mucosa, and in its midportion there was a perforating peptic ulcer 6 mm. in diameter.

Postoperatively, gastric suction was continued and the patient received intravenous fluids, electrolytes, and antibiotics. Within 48 hours the infant was passing stool, and he was given a formula on the following day. The patient was eating well and having normal stools when dismissed on the ninth postoperative day.

CASE 3.—A 2-year-old girl was admitted to the pediatric service on Oct. 21, 1952, with a 12-day history of cramping abdominal pain, fever, vomiting, and obstipation which had failed to respond to antibiotics. Two days before arriving here, she had been hospitalized at home for parenteral administration of fluids.

Physical examination on admission revealed an acutely ill, dehydrated child with a rectal temperature of 103.8 F. and a pulse rate of 120. The abdomen was markedly distended and

Fig. 2.—Multiple gas-filled loops of bowel with fluid levels.

tympanitic, and there was an occasional high-pitched peristaltic sound. The rectal examination was negative.

Laboratory studies, including urinalysis, hemoglobin, erythrocyte count, leucocyte count and urea, were normal. The differential leucocyte count showed an increase in polymorphonuclear band forms; chlorides were 76.6 mEq., carbon dioxide 17.1 mEq. and potassium 3.5 mEq. X-rays of the abdomen revealed a distended loop of small bowel consistent with intestinal obstruction (Fig. 3).

After hydration and correction of electrolyte deficiencies, exploration was carried out by one of us (Chance) through a right upper quadrant transverse incision just above the umbilicus. There was a large Meckel diverticulum with a broad attachment to the umbilicus, and near the ileum there was a perforated peptic ulcer with a small abscess localized by omentum and several loops of small bowel. Above this point the bowel was markedly distended, while below it the bowel was completely collapsed. The abscess was evacuated, the diverticulum with the ulcer was excised, and the ileum was closed transversely. The diverticulum measured 6 by 3 by 2.5 cm. and contained gastric mucosa in its distal half.

Postoperatively the patient was passing stool within 24 hours, and a diet was started in 48 hours. When dismissed on the 11th postoperative day, she was eating normally and having normal stools.

GROUP 2

CASE 4.—A 47-year-old housewife was admitted to the medical emergency service on April 14, 1947, with a six-week history of recurring bouts of cramping abdominal pain associated with abdominal distention, nausea, and occasional vomiting. Two weeks before the onset of symptoms the patient had undergone a bilateral salpingo-oophorectomy and total abdominal hysterectomy for a Grade 1 adenocarcinoma of the fundus.

Physical examination disclosed moderate dehydration, slight abdominal distention with high-pitched peristaltic sounds, and a dilated loop of bowel which could be felt through the rectum on the right. Laboratory investigations included urinalysis, hemoglobin, and erythrocyte and leucocyte counts, all of which were normal. X-rays of the abdomen demonstrated dilated loops of small bowel with some gas in the colon.

Fig. 3.—Distended loop of small bowel.

On a program of long-tube decompression and hydration the patient improved. A roentgenogram of the colon was reported as within normal limits, but dilated loops of small bowel with fluid levels were present and when cramping abdominal pain again developed, exploration was carried out by Dr. Virgil Counseller on April 21, through a secondary low midline incision.

There were a few adhesions from the previous operation, but the site of the obstruction was a Meckel diverticulum, the distal end of which was attached to the mesentery by a fibrous cord forming an internal opening through which a segment of ileum had prolapsed. The prolapsed segment had then twisted on its pedicle, producing strangulation. Nineteen centimeters of ileum with the 4 by 2.5 cm. Meckel diverticulum was resected, and an end-to-end ileoileostomy was performed. Convalescence was uneventful, and the patient was able to leave the hospital on the 12th day. When last seen at the clinic on the 23rd postoperative day, she was asymptomatic.

CASE 5.—A 24-year-old housewife, five months pregnant, was admitted to the medical gynecologic service at St. Marys Hospital on March 2, 1946, with a 32-hour history of cramping abdominal pains, nausea, and vomiting. At the time of admission her temperature, pulse, respiration, and blood pressure were normal. The only positive findings were minimal abdominal tenderness with slight rebound tenderness and the uterine enlargement consistent with a five-

month pregnancy. Laboratory studies revealed a normal urinalysis, hemoglobin content of 12.9 gm. per 100 cc., leucocyte count of 5,600, with a differential count that showed 9 lymphocytes, 2 monocytes, 27 segmented and 62 band polymorphonuclear leucocytes per 100.

Under observation in the hospital the patient became rapidly worse with cramping abdominal pains, increasing abdominal distention, tenderness, and tachycardia. The leucocyte count rose to 19,200 within 24 hours, and a shock-like picture developed. A roentgenogram of the abdomen revealed dilated loops of small bowel, and emergency exploration was carried out by Dr. J. H. Pratt on March 3. Through a primary midrectus muscle-splitting incision there was found a Meckel diverticulum, with a fibrous cord attaching the distal end to the superior surface of the mesentery, forming an internal opening through which the terminal ileum had prolapsed. The bowel was strangulated. A resection with a side-to-side ileoascending colostomy was performed. The pathologist reported a Meckel diverticulum 6 by 2 by 2 cm. with a large area of gastric mucosa and a nodule of pancreatic tissue 3 by 3 by 2 cm. in its tip. The pancreatic tissue was associated with two small areas of fat necrosis. The ileum was gangrenous with thrombosis of the veins in the mesentery.

The day following operation the patient aborted, but otherwise her convalescence was uneventful. She was dismissed from the hospital on the 13th postoperative day and from the clinic on the 19th day.

Case 6.—A 1-month-old boy was well until 24 hours before admission on Nov. 8, 1950, when he passed a bloody stool, followed by cramping abdominal pains and vomiting. On physical examination the rectal temperature was 100 F., and the abdomen was markedly distended, with a bowel pattern and visible peristalsis. There was blood in the stool on rectal examination. Urinalysis, hemoglobin content, erythrocyte count, and leucocyte count were all normal. A roentgenogram of the abdomen showed multiple distended loops of small bowel with no gas in the colon.

Exploration was carried out as an emergency procedure by Dr. George Hallenbeck. The site of the obstruction was found to be a fibrous cord from the tip of a Meckel diverticulum to the root of the mesentery, forming an internal opening through which several loops of ileum had prolapsed and strangulated. The cord was severed, and normal color returned to the bowel. Because of the patient's poor condition, the diverticulum was not removed. The infant convalesced uneventfully and was dismissed from the hospital on the 11th day. When seen at the clinic for a return visit on the 24th day, the child was eating normally and gaining weight.

Case 7.—A 3-week-old boy was admitted to the pediatric service on Sept. 2, 1951, with a 40-hour history of vomiting, increasing abdominal distention, cessation of stools, and listlessness. On physical examination the temperature was normal, the abdomen was markedly distended with visible loops of bowel, and there was an irregular soft mass in the right lower quadrant. Bowel sounds were hypoactive, and there was bright-red blood in the stool on rectal examination. Urinalysis, erythrocyte count, leucocyte count, and differential count were normal. The concentration of hemoglobin content was 18.6 gm. per 100 cc., and a roentgenogram of the abdomen revealed distended loops of small bowel consistent with intestinal obstruction.

Exploration was performed as an emergency by Dr. H. H. Belding. Through a midrectus muscle-splitting incision the patient was found to have a Meckel diverticulum with a fibrous band to the root of the mesentery, forming an internal opening through which had prolapsed the terminal ileum which was strangulated. Releasing the bowel did not restore the circulation, and a right hemicolectomy with a side-to-side ileotransverse colostomy was performed. The Meckel diverticulum was removed along with 25 cm. of terminal ileum and the right colon.

Except for a superficial wound infection the convalescence was uneventful, the patient being dismissed from the hospital on the 14th day and from the clinic on the 19th day. At a subsequent visit to the clinic, when 14 months old, the child appeared well and was gaining normally.

Group 3

Case 8.—A 7-year-old boy was admitted to the pediatric service on June 3, 1951, with a two-month history of recurring bouts of cramping abdominal pain, distention, nausea, and vomiting. During the first attack he had undergone appendectomy elsewhere without altering the course of his illness. A Miller-Abbott tube would always relieve the acute attack, but when

the eighth attack developed the patient was referred here. Physical examination on admission was normal except for moderate distention of the abdomen with visible peristalsis and audible borborygmi. Laboratory studies, including urinalysis, hemoglobin, erythrocyte count, leucocyte count, differential count, and blood sugar, were normal. A roentgenogram of the abdomen disclosed a number of gas-filled loops of small bowel in the midabdomen. The abdomen was successfully decompressed, and the patient had a five-day preparation of the colon with saline laxatives, irrigations, and 750 mg. of chlortetracycline (Aureomycin) daily. On June 21, exploration was performed by one of us (Waugh) through a secondary right midrectus muscle-retracting incision. The patient was found to have an inverted inflammed Meckel diverticulum with marked secondary inflammation of the adjacent bowel. Twelve centimeters of ileum with the Meckel diverticulum was resected, and intestinal continuity was restored by means of an end-to-end aseptic ileoileostomy over a three-bladed clamp. There was a linear ulcer 4 by 1 cm. in the diverticulum, but no gastric mucosa was found. After an uneventful postoperative course the patient was dismissed from the hospital on the 10th day and from the clinic on the 16th day.

CASE 9.—A 4-month-old boy was admitted to the pediatric service on Jan. 16, 1951, with a 48-hour history of cramping abdominal pain and vomiting. The home doctor had given an enema and noted bloody mucus in the returns. Positive physical findings on admission were moderate abdominal distention, high-pitched obstructive-type bowel sounds concomitantly with cramps, a mass extending from the right lower quadrant to the midepigastrium, and bloody mucus on the rectal glove. Urinalysis was negative, and hemoglobin content was 12.8 gm. per 100 cc. Intestinal obstruction secondary to an acute intussusception was suspected, and exploration was carried out as an emergency by Dr. George Hallenbeck. An ileocolic intussusception with a Meckel diverticulum as the leading point was found and reduced, but the ileum in the region of the diverticulum was gangrenous. Twenty centimeters of ileum, with the inverted Meckel diverticulum measuring 2 by 1.5 by 1.5 cm., was resected, and intestinal continuity was restored by means of a side-to-side ileoileostomy. The patient did well postoperatively and was dismissed from the hospital on the 13th postoperative day. A return visit at the clinic on the 29th postoperative day disclosed that the child was eating and gaining normally.

COMMENT

Attention was first directed to ulceration as a complication of Meckel's diverticulum by Denecke in 1902. In 1904 Salzer first reported the presence of gastric mucosa in a Meckel diverticulum, but it remained for Deetz, in 1907, to point out the relationship of gastric mucosa to ulceration in a Meckel diverticulum. Once attention had been called to the complication of ulceration in a Meckel diverticulum, many reports began to appear in the literature. Jackson † was first in this country to report a case of intestinal hemorrhage from ulceration in a Meckel diverticulum, and the relationship of pain to the taking of food was noted by Dragstedt.

By 1936, Cobb was able to collect from the literature reports of 98 cases of peptic ulcer in Meckel's diverticula, to which he added 2 of his own. Of this group 72% had intestinal hemorrhage and 55% had perforation, but no mention was made of intestinal obstruction. Among the first reports of intestinal obstruction secondary to ulceration related to Meckel's diverticula was that of Brown and Pemberton of two cases of partial obstruction. Since these diverticula did not contain gastric mucosa, the authors thought the relation of the ulcers to the diverticula incidental. Waugh, Herrell, and Crumpacker were the first to report intestinal obstruction as a result of cicatrization and inflammatory reaction associated with active ulceration, similar to that in peptic ulcer, in or near Meckel's diverticula. In both cases the diverticula were found to contain gastric mucosa with typical parietal cells.

† References 9 and 10.

It is now known that gastric mucosa is found in a high percentage of the diverticula which develop complications that require surgical treatment. In the majority of cases these will occur in the early years of life, but they may occur at any time. Gastric mucosa was present in 54% of 130 cases reported by Gross and in all but 1 of 31 cases of symptomatic diverticula reported by Freedman and his associates. Since the incidence of ulceration and perforation is high in these patients, we believe that undoubtedly many with a localized inflammatory process in the peritoneum must present with the clinical picture of an acute intestinal obstruction, but this has not been clearly indicated in the literature.

The three cases comprising Group 1 all presented with a clinical picture of a mechanical intestinal obstruction which was secondary to a localized peritonitis which arose from complications in the Meckel diverticula. In two cases this was due to the perforation of a peptic ulcer in the diverticulum, while in the third the perforation was probably the result of diverticulitis. The localized inflammatory process by compression, angulation, and production of a regional ileus resulted in a complete mechanical obstruction of the ileum. Edema of the bowel wall may also contribute to the obstruction but is not an important factor in the acute phase, as may be seen by the fact that in two of the cases evacuation of the localized abscess and removal of the diverticulum with transverse closure of the ileum was promptly followed by relief of the obstruction.

That intestinal obstruction could result from an adhesive band, as it did in the four cases in Group 2, or from an intussusception, as it did in the two cases in Group 3, has long been known. Indeed, until ulceration was pointed out as the leading complication in Meckel's diverticula, obstruction from an adhesive band was the chief cause of reported complications. In the 326 cases of surgically treated Meckel's diverticula collected by Wellington, 212 had obstruction—144 by adhesive bands, 59 by intussusception, and 9 by volvulus.

The origin of this adhesive band, which is the terminal ligament, has been much discussed in the literature. It was recognized by Meckel,‡ who pointed out that it was the remains of the omphalomesenteric artery and vein which were transformed into a fibrous cord. This fibrous cord may be attached to the umbilicus, mesentery, or intestine. Most commonly it is attached to the mesentery, thereby forming an internal hernia through which bowel may pass and become obstructed. When attached to the umbilicus alone, it rarely, if ever, is the site of obstruction, for there is no internal hernia. Inflammatory adhesions between the diverticulum, or its terminal ligament, and neighboring organs so as to form an internal hernia rarely occur.

In all four cases in which the obstruction was produced by an adhesive band, the attachment was to the mesentery.

Surgical treatment in these nine cases consisted of excision of the diverticulum and transverse closure of the ileum in two cases, resection of a portion of ileum, or ileum and colon, followed by end-to-end or side-to-side anastomosis in six cases, and excision of the obstructing fibrous cord in one case. There were no operative deaths or serious postoperative complications in this group of nine cases.

‡ References 12 to 14.

SUMMARY

Nine cases of intestinal obstruction secondary to Meckel's diverticula are reported.

The manner in which the obstruction was produced put the cases into three groups. The first, three cases, was secondary to a perforation with a localized inflammatory process; the second, four cases, was secondary to a fibrous cord forming an internal hernia, while in the third, two cases, the diverticula were the leading point of an intussusception.

Surgical treatment consisted of excising the diverticula with transverse closure of the bowel in two cases, resection of a portion of bowel with immediate anastomosis in six, and excision of the fibrous cord thereby freeing the bowel in one case.

There were no operative deaths.

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PITFALLS, PRECAUTIONS, AND COMPLICATIONS IN CARDIAC RESUSCITATION

Study of 1,200 Cases

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THE PURPOSE of this paper is to call attention to complications likely to be encountered and to warn of pitfalls in attempts to resuscitate the acutely arrested heart. Unless one is familiar with some of the hazards, the golden opportunity to restore the normal heart beat may be lost.

Since cardiac arrest is a relatively infrequent experience for any particular surgeon, it is likely that a surgeon's personal observations will be insufficient. This report is based on a study of over 1,200 cardiac arrest case reports sent to us through the cooperation of physicians in all parts of the United States, as well as foreign countries. The following comments are based on our actual experiences and on the experiences of those contributing to this survey.

DIAGNOSIS

Pitfalls in the diagnosis in cardiac arrest should be minimal. With an absent palpable pulse and no blood pressure, the burden of proof would seem to rest with him who says cardiac activity is sufficient for life. Actually, however, more patients lose their chance for recovery during this crucial period between arrest and diagnosis than from any activity in the resuscitative period. As we have emphasized on previous occasions, the time factor represents the crux in successful resuscitation. Figure 1 dramatically illustrates that the hand must be on the arrested heart and prepared to massage within four minutes of the arrest. In 94% of 1,200 cases in which the results were considered successful, the heart was massaged within the first four minutes. Only 6% survived after a delay of over four minutes. One editorial on cardiac arrest recently stated that "if the interval was between 5 and 15 minutes, the recovery rate fell to 33%." Of the 6% that survived a delay of

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over four minutes in our series of 1,200 patients, the majority were under five minutes. A 33% recovery rate with this degree of anoxia is not possible under present conditions.

The importance of the time element in the treatment of this condition must always be kept in mind, for a period of reversibility is present in almost all cases of cardiac arrest. To allow this period to pass in needless diagnostic procedures, in the useless administration of various stimulants, in the frantic search for a stethoscope, or in obtaining consultation from others, is to allow a life to be lost

TIME
FACTOR
94% OF SUCCESSFUL CASES MASSAGED WITHIN 4 MINUTES. (1200 CASES)
ONLY 6% SURVIVED AFTER A DELAY OF MORE THAN 4 MINUTES.

Fig. 1.—Cardiac arrest is essentially a three- to four-minute emergency. Irreversible damage is likely to occur if a delay of more than four minutes is made before intermittent cardiac compression is begun.

that otherwise might be saved. The problem often is as simple as that. It is practically useless to seek help in resuscitation unless immediately available.

We agree with Gordon Murray [10] when he recently urged that the diagnosis of cardiac arrest, with subsequent attempts at cardiac resuscitation, be made in many cases of drowning, accidental electrocution, strangulation, anoxia, and poisoning from noxious fumes and gases.

Pitfalls in diagnosis, unfortunately, are sometimes due to an anesthetist's trying to take care of two rooms at once or simply due to a lack of the proper alertness required. All too frequently our attention is called to a case in which the surgeon

makes the diagnosis by the absence of bleeding in the wound or other signs of an inadequate circulation. We recall one case in which a visiting surgeon entered the operating room and was complimenting the operator on the hemostasis achieved, when actually the patient was in cardiac arrest! After an emergency thoracotomy, the heart quickly returned to a regular rate, but the patient, a 35-year-old robust man, died some hours later of the prolonged anoxia experienced prior to the diagnosis, which should have been made promptly.

Several times we have been asked how often a mistaken diagnosis has occurred. We are aware of three such cases, although doubtless there are a number. During an exploratory laparotomy for carcinoma of the stomach on the Fourth Surgical Division at Bellevue, the patient's blood pressure suddenly fell to zero. The aorta had no pulsation. The chest was quickly opened, and just after this procedure the heart returned to spontaneous activity. The patient's postoperative course was not complicated by the thoracotomy. In the two other cases, the heart was found to be beating so feebly that cerebral circulation was probably inadequate and massage

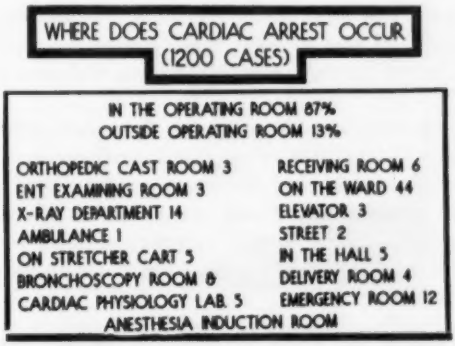

Fig. 2.—Chart showing that cardiac arrest may occur at almost any place in the hospital. It is not wholly an operating-room emergency.

was beneficial. In both cases the patients recovered. The fear of an occasional mistaken diagnosis should not be a deterrent.

WHO SHOULD ATTEMPT RESUSCITATION?

One author answers the question of who should attempt resuscitation by suggesting that each hospital have an emergency cardiac team. Such a plan, however, would be doomed from the start, for it fails to consider the crux of the whole problem—the time factor. Obviously, a group of persons can not be assembled rapidly enough even under the most optimal conditions.

The question resolves itself to one answer. The person to attempt cardiac resuscitation is the person making the diagnosis, for it is with this person that the patient's chances of survival rest. At Bellevue Hospital during the past three years, particular emphasis on cardiac resuscitation has been centered about the intern, since it is most frequently the intern who is with the patient in the induction room, in the elevator, on return from the operating room, or in the patient's room in the postoperative period. It is the intern who most frequently will have the optimal chance to attempt resuscitation in those cases occurring outside of the operating room.

Certainly, knowledge of resuscitation should not be limited to the thoracic or the general surgeon. Figure 2 illustrates the need for every physician's being familiar with proper methods for resuscitation. We know of a pediatrician, several anesthesiologists, and internists who have successfully attempted resuscitation. We are also familiar with many tragic cases in which the physician had not felt it necessary to acquaint himself with this technique. This would include ophthalmologists, plastic surgeons, general surgeons, obstetricians, radiologists, and even cardiologists. Short courses are now being given in several centers, such as our own, where the physiological and pharmacological, as well as practical, aspects of resuscitation may be learned. As Dr. Beck has stated, "No surgeon should be allowed to operate tomorrow if he is unable to resuscitate a heart today."

THE INCISION

Although the standard fourth left anterior interspace seems to provide the most adequate approach for manual massage of the heart, a great many variations are attempted. Recently, one of us saw an autopsy on a patient who died of sudden cardiac arrest in one of our large teaching centers. The incision was placed in the second left interspace and was so short that certainly no more than two fingers could be inserted. Cardiac massage represents a great deal more than a mere tickling of the heart!

Frequently the operator will fail to realize the speed with which the incision should be made and will take time to cut costal cartilages, remove a rib, or insert a rib spreader, all of which add vital seconds at a point where irreversible cerebral cortical damage can occur. The cartilage can be cut or the rib spreader can be inserted at a later point. In 57 personally experienced cases, we have yet to see an incision which could not be spread manually in order to allow the hand to be inserted. Figure 3 illustrates this.

An incision made too medially has resulted in the death of at least four patients. Such an incision cut the left internal mammary artery. On closure of the chest the bleeding point was not controlled. A wide incision is worth while, however, but should be extended in the lateral direction, as pictured in Figure 3.

In two cases, perhaps the most drastic of complications occurred. The operator, impressed with the importance of speed and bold action, made the incision with such intensity that when the chest was spread the heart itself was found to have been almost severed! The incision should take only 10 to 15 seconds, since there is no bleeding; the only pause need be made at the point of entry into the pleura. The left lobe of the lung has been lacerated on occasion, but can be repaired with little difficulty.

Although we personally have not encountered any such cases, we are aware of three cardiac arrests which occurred to patients in body casts! In describing one of these cases, Julian Johnson suggests that it be mandatory that a window be made in body spicas allowing for cardiac massage, if necessary.

Articles continue to appear advocating various approaches to the heart in cardiac arrest. Many of these are designed to avoid entry into the pleura and are made subcostally.[17] Some of these same approaches have been advocated by others over 40 years ago. The thoracic approach would seem, however, to have many advantages not offered by the other methods. Not only does it provide a rapid and easily accessible approach, but it allows direct visualization of the heart. Unless this is

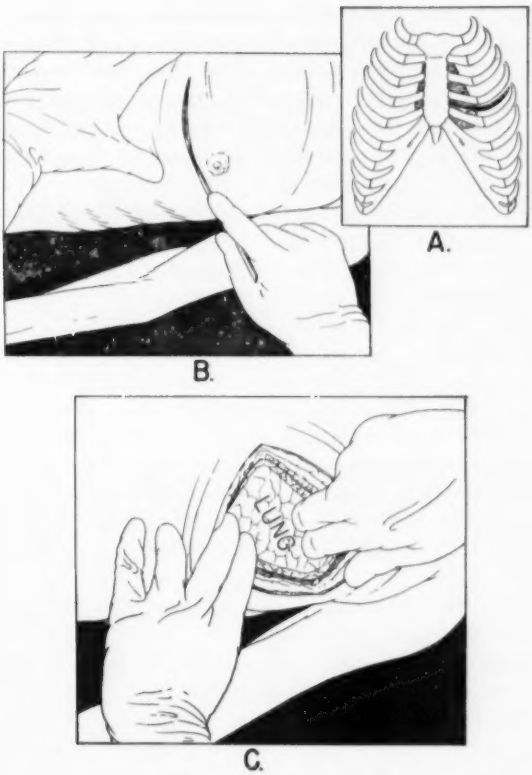

Fig. 3.—*A*, optimum approach is through the fourth left anterior interspace as shown. Caution against carrying the incision too far medially should be made. *B*, a rapid incision can be made in the chest wall in a matter of several seconds. The incision should be carried at least as far as the anterior axillary line. *C*, adequate exposure can be obtained for introduction of the hand by spreading the ribs as pictured. A rib spreader may be introduced later if necessary.

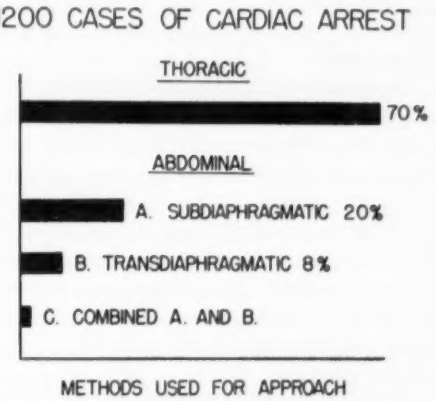

Fig. 4.—The majority of attempts at cardiac resuscitation in this country have been by the transthoracic route. The abdominal approach, however, is still used in some parts of the world.

possible, the heart activity cannot easily be evaluated. Ventricular fibrillation is not easily recognized. The thoracic exposure makes the injection of drugs a less hazardous procedure, and the application of the electrodes is relatively simple. The pericardial sac can be opened, if necessary. Experimentally, no other approach has shown evidence of making possible as great a cardiac output. Shunting more blood into the cerebral or coronary circulation by manually clamping the arch of the aorta beyond the great vessels provides another advantage.

Figure 4 gives the distribution for the approach used in 1,200 cases.

Norbury [11] states that the thoracic route was the first method used in cardiac resuscitation. Even though the abdomen was open at the time, Ingelsrud, in 1901, performed an emergency thoracotomy. Starling and Lane [13] successfully resuscitated their case in 1902 via the subdiaphragmatic route.

Most of the objections to the thoracic route were because of the resulting pneumothorax. Frazier,[8] in 1911, summarized the results with the various methods and concluded that the subdiaphragmatic route was best, with 10 good results out of 16 cases and only 7 favorable results out of 24 cases in which the thoracic route was used. He concluded that because of the elasticity of the chest in children one could make adequate pressure on the heart without opening either chest or abdomen. It is probably true that a rare case will respond to mechanical stimulation if it is given almost at once. One physician several years ago reported such a case revived by fracturing one of the ribs.

With the transdiaphragmatic incision, one surgeon recently encountered intractable bleeding from the heart.

Another method used was that of turning a flap of chest wall to expose the pericardium and avoid causing a pneumothorax. Two years ago we saw a chest that had been entered by an incision made at right angles to the ribs! In 1904 Keen [8] described a method of massage with one hand on the chest and the other under the diaphragm. Mauclaire was perhaps the first to suggest the transdiaphragmatic route (1902). An extraperitoneal modification of the abdominosubdiaphragmatic route was proposed by Tomaselli in 1907.

MASSAGE

If any doubt exists of the value of adequate cardiac massage in maintaining adequate blood flow, then the following experience should be most convincing. One of us recently was introduced to a middle-aged patient who had experienced a cardiac arrest while undergoing an orthopedic procedure under spinal anesthesia. The patient quite vividly recalls having regained consciousness during the period of massage and having looked up at the reflecting mirrors overhead and observed the surgeon's hand in his chest! Needless to say, he was quickly anesthetized with a general anesthetic agent. The patient denies having experienced any pain sensation.

The commonest pitfall in the actual intermittent compression of the heart, which we term "massage," lies in the inability of the operator to massage in a fashion that is adequate for oxygenation of the cerebral cortex. This is usually a result of his failure to have practiced the procedure in the animal laboratory or on suitable models. (Such a model was available at the 1953 Annual Meeting of the American Medical Association.)

Charles S. White,[18] in 1909, suggested that "heart massage, *herz-massage,* or *massage de coeur* in its surgical meaning, implies the manual stroking, rubbing, or

kneading of the organ, either by costal resection, through an incision in the diaphragm or with the diaphragm interposed between the hand and the heart." Actually, cardiac massage has now come to imply more of a left ventricular compression which should be of sufficient intensity to allow a palpable pulse in the peripheral vessels, maintain adequate-appearing coloration of the skin, and keep the pupils constricted. In the animal laboratory, as well as clinically, these criteria are more easily met if a relatively rapid rate of massage is maintained, i. e., 60 to 85 strokes a minute. Figure 5 illustrates the position of the hand which we find best for adequate massage. It is not necessary to relax and allow the heart to fill between compressions as is so often recommended.

Keen gives Schiff credit for having introduced manual massage of the heart, in 1874. Like other aspects of resuscitation, a number of methods of massage have been suggested, such as the two-hand method, compression of the heart against the sternum, and massage by the use of suction cups. In our hands, we find the method as pictured the optimal one from a practical as well as physiological standpoint.

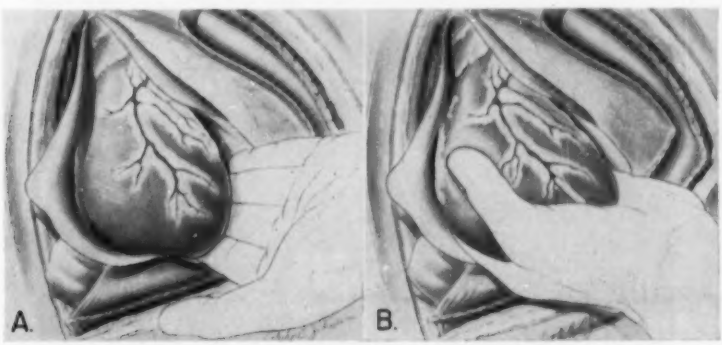

Fig. 5.—*A*, introduction of hand for manual massage; *B*, massage of heart must be such that a blood pressure of 60 to 70 mg. is produced, that the pupils remain constricted, and that the patient remains well oxygenated.

It is of importance to recognize that the initiating cause of the cardiac standstill has nothing to do, as a general rule, with the failure of spontaneous beats to recur on massage. The ventricular specific tissue is the least vulnerable to oxygen lack on the basis of forming adequate stimuli. It should be remembered that the human specific tissue can be made to function 60 hours after death if placed under suitable conditions. The myocardium, however, is most vulnerable to oxygen lack, and it is because of this that spontaneous beats do not occur because of the failure of the metabolites to be moved.

How long should one massage? The beginner is likely to be discouraged too early. Often a flabby and atonic myocardium can be restored to an actively beating organ. Our most surprising return of active rhythmical cardiac action occurred after two hours of massage. Hosler [4] mentions the case of M. N. Foote which was successfully resuscitated in 1950 after eight hours of manual cardiac massage. The patient returned to the active practice of law. Our rule of thumb is to continue massage as long as we are convinced that artificial cerebral circulation has been adequate enough to prevent irreversible damage of the cortical cells.

PERICARDIUM

Should the pericardium be opened in every case of cardiac arrest? Some have advocated a routine incision of the pericardial sac. The disadvantages of opening the pericardium include (1) delay in massage if done just after opening the chest; (2) addition of increased technical difficulty for those unfamiliar with procedure—vagus or phrenic nerve may be severed; myocardium may be lacerated; (3) trauma from massage is appreciably greater as noted on autopsy specimens, and (4) complications from inadequate closure of the pericardium may result. Too tight closure

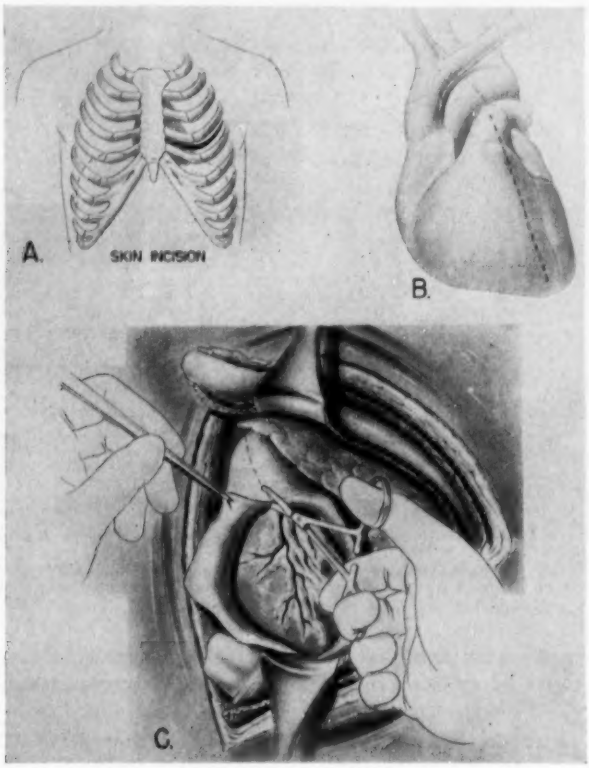

Fig. 6.—*A*, diagram showing skin incision just below the nipple and usually before the fourth and fifth ribs on the left. *B* and *C*, pericardium may be incised in the fashion shown in diagrams *A* and *B*. In the majority of cases, opening pericardium will not be necessary.

allows collection of serosanguineous exudate, resulting in cardiac tamponade. Herniation of a portion of the heart with a resulting tamponade effect has been reported. We suspect that the average heart can be compressed in a more adequate fashion when lying within the pericardial sac.

It will be necessary to open the pericardium on occasion (Fig. 6). In elderly persons with a thickened pericardium, it is not always possible to be sure that ventricular fibrillation is not present until the pericardial sac has been opened. In the young pediatric case, we have found it necessary to open the pericardium because

of the size of the heart and the diaphragmatic attachment of the pericardial sac. For the application of the electrodes in ventricular fibrillation, we have always opened the pericardial sac.

RUPTURE OF THE HEART

Rupture of the myocardium of the heart during manual massage of the heart, which occurs more frequently than is reported and is a complication of the resuscitative procedure, is to be avoided. Its occurrence almost always adds disaster to an already serious catastrophe. In the cases which we have experienced or studied,

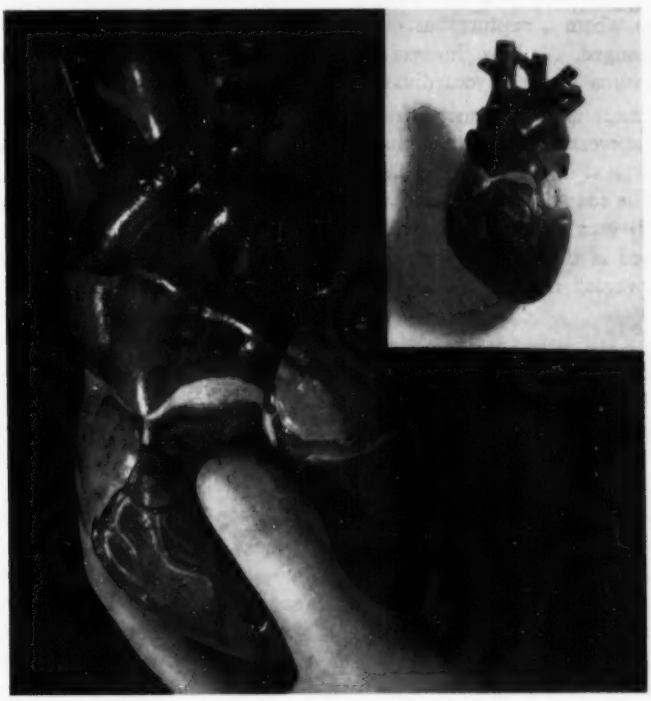

Fig. 7.—Rupture of the heart is not an infrequent occurrence. Its commonest site is, as pictured in the inset, where the circle indicates the point of rupture. This is usually due to prolonged pressure with the thumb and is usually a complication of resuscitation in patients with previous myocardial disease.

rupture of the heart has occurred nine times. Hurwitt and Seidenberg recently reported a case. Figure 7 illustrates that area of the heart most likely to be ruptured. Rupture most commonly is due to prolonged, localized, and excessive pressure on the myocardium by the thumb. The anterior wall of the right ventricle may already be weakened by preexisting cardiac pathology in the elderly, such as excessive myocardial fibrosis, old myocardial infarct, and coronary arteriosclerosis. In all but one of the cases, the pericardium had been opened.

In a case that one of us encountered during cardiac resuscitation on one of the wards at Bellevue Hospital, a rupture occurred but was small enough that a fatal

result was not immediate. The patient's heart was resuscitated, but cortical damage was too severe and death resulted in 12 hours. Awareness of this complication may help to prevent its occurrence.

Opening the pericardium adds to the danger. With the intact pericardium, massage is less traumatic and allows the pericardial fluid to act as a lubricant as well as to prevent continued pressure on the exact spot at all times. Pressure should be more evenly distributed between the palm, the fingers, and the finger tips. Massage should never be continued too long in one spot without shifting the position of the hand a bit. Additional care should be taken in the case with arteriosclerotic coronary vessels or with known previous heart disease. The youngest patient in whom a rupture has occurred was 52 years old. Massage had usually been prolonged. We have, however, massaged hearts as long as two hours without severe trauma to the myocardium being apparent.

Treatment is seldom successful. The one case that we encountered is the only partially successful one that we know of. A more difficult problem presents itself than is present in the closing of the left ventricle in aortic valvulotomies. Attempts to close the rent by suture usually only enlarge the size of the hole because of the friable adjacent tissue. The anterior descending coronary artery may inadvertently be included in the suture. Absorbable gelatin sponge, as described by Jenkins and his co-workers,[6] may be of some value in the smaller rents.

INTRA-ARTERIAL TRANSFUSIONS

In spite of several recent reports demonstrating the effectiveness of rapid intravenous administration of blood, the intra-arterial transfusion route remains the most effective in specifically indicated cases of cardiac arrest. The advantages of the intra-arterial route have been reviewed by Stephenson and Hinton.[14]

The usual case of cardiac arrest seldom will require the rapid administration of whole blood. It will be those cases with diminished blood volume and poor cardiac refill, even with massage, which will respond to rapid administration of blood. With intra-arterial transfusion, whether it be administered directly into the aorta,[14] the femoral artery, or a peripheral artery, the rapid flow of blood will enter the aorta in a reversed direction, pushing the aortic valves tight and shunting off into the coronary and cerebral circulation. Complications of intra-arterial transfusion can be major ones, including death as the result of allowing an air bubble in the stream of blood, gangrene of the extremity distal to the transfusion, and overadministration of blood. Because of the delay of cutting down on a peripheral vessel such as the radial artery, the femoral or aortic route seems most desirable. When cardiac arrest occurs in the presence of severe blood loss (such as breakdown of a resected coarctation anastomosis), all peripheral veins are collapsed and will require a cut down to find an adequate one. This, too, takes a great deal more time than simply inserting the needle into the readily available aorta.

CLOSURE

The closure will be much the same as in any thoracic incision. Provision for underwater drainage should be carried out, and we practice the administration of 2 gm. of streptomycin and 600,000 units of penicillin injected in the pleural cavity before closure.

INFECTION

Since the luxury of sterile precautions in cleansing the chest and draping the incision can seldom be afforded in this emergency, it is logical that some concern should be expressed about the danger of empyema, pericarditis, mediastinitis, and cutaneous infections. There has been, however, a striking absence of such infections following thoracotomy for cardiac massage, even when the operator is unable to use gloves and other sterile precautions. Many infections are probably prevented by the practice of injecting penicillin and streptomycin into the thoracic cavity before closure and inserting a rubber tube with underwater drainage for several days. Occasionally, the chest may have to be tapped to remove a small collection of fluid. It is therefore important that considerations relative to a possible infection be disregarded if they are to contribute to any initial delay in instituting cardiac massage.

A note of precaution to the operator—prolonged massage will frequently result in an abrasion of the skin of the back of the left hand. One of us (H. E. S., Jr.) has had two minor infections resulting from prolonged pressure of the hand against the rib cage during the manual intermittent compression of the heart.

OXYGEN SYSTEM

All too frequently the attention is centered on effort directed at the heart, with little regard to artificial respiration. Unless a proper understanding and knowledge of artificial respiration is present, all attempts at massage of the heart will be futile. Should the cardiac arrest occur in the operating room, the anesthetist will be available and the artificial respiration can be adequately managed. In almost 15% of cardiac arrest deaths, an anesthetist will not be present and artificial respiration must be handled by the available equipment. Mouth-to-mouth breathing has been used with some success. With the use of the mobile cardiac resuscitation unit,[15] we feel we have solved this problem satisfactorily in that the unit is equipped with a positive-negative pulmonary resuscitation unit which fills the alveoli to any desired pressure as well as withdraws the air from the lungs. In spite of proper equipment for resuscitation, failure may occur if daily check of the machine and its equipment is not carried out. The positive-negative pulmonary resuscitator can be applied with the use of a face mask unless an endotracheal tube is not immediately available. Unless one sees the lungs expand and contract through the open chest, one knows that the airway is obstructed. The resuscitator will also give a warning sound. Most frequently the obstruction is due to the tongue that has dropped posteriorly, and this can be corrected with an airway or by grasping the tongue with a towel clamp and pulling it forward.

VENTRICULAR FIBRILLATION

The principal failures in achieving success in ventricular fibrillation result from (1) lack of an electrical cardiac defibrillator (such equipment should be minimal standard equipment for any hospital); (2) failure to recognize ventricular fibrillation when present, and (3) lack of experience with the technique of defibrillation. The surgeon may have difficulty distinguishing between fibrillation and standstill after a thoracotomy has been performed and the heart is in his hand because the fibrillation is weak or it is not seen through the pericardium.

Once cardiac massage has been started and ventricular fibrillation is present, there remains little need for urgency in the resuscitation procedure. Not long ago, one of us rushed by ambulance from the lower east side of Manhattan to the upper west side in order to defibrillate a heart at a hospital which did not have an electrical defibrillator. In spite of the prolonged massage in the interval, electrical shock was successful. The patient survived nine days. Almost every heart can be defibrillated. The heart of a dog can be defibrillated even when completely removed from the animal and without the presence of any circulating blood.[16]

The danger from explosion with the use of the electrical defibrillator is almost nil. The chest will have been open for a period of minutes, and all anesthetic mixture will have been discontinued prior to the thoracotomy. Diffusion of the gas mixture should be adequate.

Unless proper defibrillation equipment is used, there is danger of the surgeon or assisting personnel receiving a shock. Makeshift defibrillators can cause serious burns, and in one case of ventricular defibrillation such a unit shorted at the time

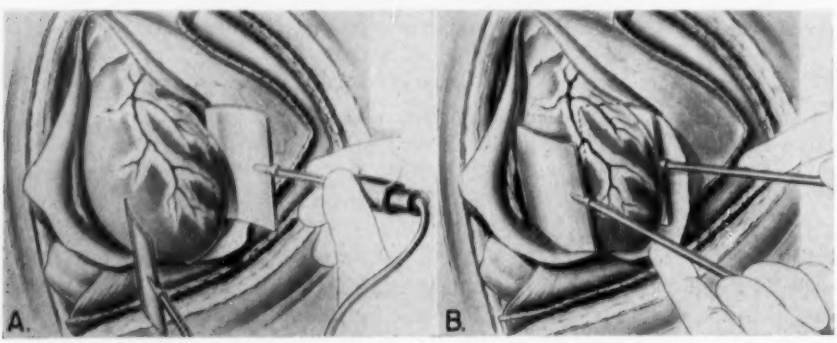

Fig. 8.—*A*, method for introducing metal electrodes for electrical defibrillation of the heart in ventricular fibrillation; *B*, method of applying moistened electrodes snugly against the heart and using electrodes which more nearly correspond to size of heart. Repeated shocks to heart given in a serial fashion may be necessary.

of its use, with subsequent death of the patient. It is helpful if the defibrillator has a transformer incorporated which will allow the anesthetist to retain a hold on the patient during the shocking procedure.

While electrical defibrillation is occurring, the patient will make a convulsive movement, throwing the arms in an upward and inward direction. It is at this point that the intravenous infusion needle may be dislodged unless care is taken.

In ventricular defibrillation we do not use the saline-moistened gauge, as recommended by some, but apply the metal electrodes directly against the heart. By applying electrodes shaped to conform to the convexity of the heart and of adequate size, one is able to get the maximum surface contact with the minimum of danger from burn. So far, we have not noted this complication with our present type of electrodes (Fig. 8).

Fortunately, from the entire group of cases, only 12% of the cardiac arrests were associated with fibrillation. It is also of interest to know that the permanent survival rate of all cases of cardiac arrest was 28%, and of those in fibrillation the rate was 29%. The heart was restored to a normal rhythm from asystole in 56%,

while in ventricular fibrillation, the heart was defibrillated in 63%. This is particularly significant when one recalls that the first proved case of ventricular defibrillation with survival occurred less than seven years ago.

USE OF DRUGS

A recent editorial in one of the surgical journals states that an intracardiac injection of procaine hydrochloride and epinephrine solution should be made in all cases of cardiac arrest as soon as the heart is exposed and massage begun. This,

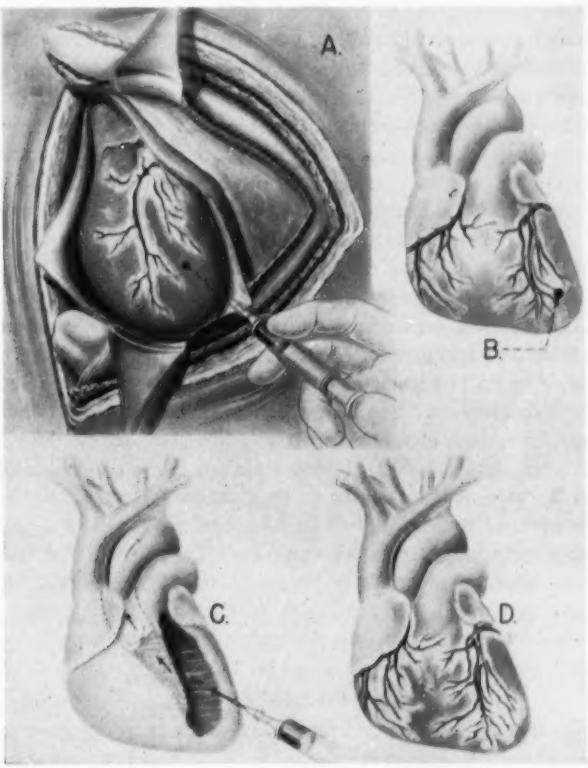

Fig. 9.—*A*, administration of drugs intracardially can best be done in this fashion and into the most readily available site of the heart. *B*, site for needle injection, being cautious to avoid coronary vessels. *C*, drugs such as epinephrine can be injected into the left side of the heart without ill effects and with a more rapid course into the coronary vessels. *D*, the sooner the drug reaches myocardium through the coronaries, the quicker will be its expected action.

we believe, to be an error in judgment, as a large number of successful cases require only manual massage of the heart before a forceful and regular rhythm returns.

Many complications and much misunderstanding has resulted from an inadequate knowledge of the pharmacology of cardiac resuscitation. For almost two years, we have been testing various drugs in the animal laboratory, and, in addition to clinical experience, are convinced that injudicious use of various recommended drugs causes great harm. With a better understanding of cardiac arrest, we are

receiving fewer cases that have received drugs such as nikethamide (Coramine), caffeine, and pentylenetetrazol (Metrazol), which are of no value in resuscitation. Those like caffeine and sodium benzoate and nikethamide are primarily cerebral stimulants and increase the metabolic demands of the already oxygen-depleted cortical cells, thereby causing further neurological sequelae or death. Procaine has been widely used in asystole with epinephrine to prevent ventricular fibrillation. Ament and his co-workers [1] regard this as standard procedure. This has been discontinued by us because of the resulting diminution of tone to the myocardium and its questionable protection against fibrillation. Epinephrine remains the most satisfactory drug for increasing the tone of the myocardium. Excessive doses should be avoided. Although calcium chloride has been advocated more recently in selected cases, we have had more success with barium chloride (2% solution), both in the animal laboratory and clinically.

Drugs need not be injected meticulously into any certain chamber of the heart. The left ventricle is the most easily accessible and the most logical route for administration, since the purpose is to get the drug into the myocardium via the coronaries as quickly as possible (Fig. 9). So far, we have not been aware of any damage to the coronary circulation from a needle injection, although it is a possibility.

In spite of the often-repeated suggestion by some authors, we have opposed the practice of first injecting epinephrine into the heart by the use of a long needle through the anterior chest wall. It is true that an occasional case of cardiac arrest will respond from the stimulus of the needle puncture. In the vast majority of cases, however, time will be spent in obtaining the syringe, filling it with solution, acquiring a proper needle, and injection—so much so that the patient's chances for survival will have been severely compromised even before effective resuscitative attempts have been started. With an absent heart beat, the epinephrine cannot be transported to the myocardium via the coronaries. Drugs given into the pericardial sac have demonstrated their ability to be absorbed,[19] but not in a period of time likely to be effective. As is true with the use of the external cardiac pacemaker, so it is with the external injection of drugs. If ventricular fibrillation is present, only a thoracotomy will allow for effective measures at resuscitation. The use of atropine has its greatest value in the prevention of cardiac arrest, rather than in the resuscitative period.[12]

EQUIPMENT

It is obvious that in such an extreme emergency as cardiac arrest all necessary equipment should be instantly available.

A simple pocket knife has been of great value in several instances and might be a worth-while instrument for the physician to carry in his pocket, rather than to wait on the more dignified scalpel—should cardiac arrest occur outside of the operating room.

Unless the circulation is almost immediately restored, some means of artificial respiration, as already mentioned, must be available.

A portable defibrillating unit should be available in all operating rooms. For those hospitals having a recovery room adjacent to the operating suite, a mobile cardiac resuscitation unit is a great adjunct. One of us (H. E. S., Jr.) has devised such a unit which has been in use on the Fourth Surgical Division at Bellevue Hospital for several years. The unit is equipped with oxygen tanks, masks, an

automatic positive-negative pulmonary resuscitator, a defibrillating unit, tracheal suction apparatus, intra-arterial transfusion unit, EKG machine (direct-writer), and syringes and drugs necessary for such an emergency. Special equipment is also included for pediatric resuscitation, whether it be respiratory or circulatory.

POSTRESUSCITATIVE CARE

The immediate postresuscitative period is frequently one of confusion. After the chest has been closed and there is satisfaction that cardiac action is adequate, spontaneous respirations may still not have returned. In cases with subsequent recovery, this may not occur for as long as 20 minutes and even longer. Artificial respiration by means of pressure on the anesthetic bag or by use of the positive-negative respiratory resuscitator will be indicated. Seldom will it be necessary to place a patient in the artificial lung. On occasion, rhythmical stimulation of either of the phrenic nerves will serve markedly to augment respirations as well as to improve venous return to the heart. For the most part, this has been a neglected aspect of the postresuscitative period.

Although cerebral stimulants have been cautioned against in the period of anoxia and cardiac arrest, it is permissible to use them at this stage. Nasal oxygen has been used by us in preference to an oxygen tent, for we have found that the patient in a tent is more likely to be neglected.

Tracheal suction should be available in all cases should return of active cerebration be delayed. Collection of tracheal secretions in the bronchial tree have been responsible for a number of deaths.

All too often the nursing care of these cases will be neglected. Those, of course, on whom massage is started promptly will need little care. The rest will need frequent turning and preventative measures against pressure ulcerations. Rehabilitation measures should be instituted as soon as the patient returns to his bed.

Protection of the eyes may be necessary in those patients having suffered a rather marked amount of cerebral anoxia. A drop of liquid petrolatum in each eye as well as a light dressing for the eye will serve to protect them.

The lower nephron syndrome may appear in certain cases due to prolonged anoxia of the kidney and may need the consultation of a competent urologist. An accurate knowledge of the urinary output should be available in all cases, and in the majority a catheter should be inserted into the bladder.

Adequate hydration can be accomplished by the intravenous administration of dextrose in water with little saline. If the patient is unable to feed himself, then a continuous-drip feeding through a catheter in the stomach or by intermittent fluid administration through a tube should be instituted. If large amounts are given at one time to the unconscious patient, the patient should have his head and chest elevated to prevent gravitational aspiration of stomach contents.

Chemotherapy should be continued for five or six days, or until the temperature is normal.

Should hyperthermia develop in the immediate postresuscitative stage, the body should be given alcohol rubs, and ice bags should be placed in the axilla and inguinal area. Of those patients who die, the majority will die in severe hyperthermia during the first 24 hours; 87% of those patients who do not survive an episode of cardiac arrest will die within the first 24 hours.

Laboratory studies should include hemoglobin determinations and a blood volume determination, if available. Roentgenograms of the chest should be taken on the second or third day. An electrocardiogram should be taken at frequent intervals to determine accurately the activity of the heart. Serial readings will often show progressive improvement with disappearance of arrhythymias. Frequently the only abnormality noticed will be a low voltage and a persistent tachycardia. Digitalization of the patient is indicated frequently.

We have used levarterenol enthusiastically after the heart action has been restored and a peripheral hypotension remains. By controlling the drops per minute, a near-normal blood pressure may be obtained.

The neurological sequelae of prolonged cerebral anoxia in cardiac arrest will be discussed in a subsequent report.

MEDICOLEGAL

One is frequently asked about the medicolegal implications in cardiac resuscitation. A number of malpractice suits of such nature have been called to our attention. None has been decided for the plaintiff. When the physician encounters a case of sudden cardiac arrest in which associated findings do not show signs of an irreversible situation, then it is obvious that he must act to the best of his judgment, disregarding any necessity for consent from relatives.

Through the lay press, the average person has become aware of cardiac resuscitations with increasing frequency. It is now a well-accepted procedure by the medical profession, for which the surgeon is responsible.

SUMMARY

Through a pooling of experience, with the cooperation of individual physicians in many parts of the country and several foreign lands, this report has been made possible. An attempt has been made to correct misconceptions and point out pitfalls and complications likely to be encountered in the resuscitation of the acutely arrested heart. Unless the physician is aware of the precautions necessary, his efforts are likely to be futile. With the reality and effectiveness of cardiac resuscitation having been well established, the responsibility for a thorough knowledge on this subject rests with each physician.

Not only does such a pooling of knowledge aid in the practical approach to the problem of cardiac arrest, but it opens new avenues of information relative to cerebral anoxia and respiratory resuscitation, as well as to the activity of the heart.

Many physicians of the United States and foreign countries have made this study possible by sending us their cases. We hope to continue this work.

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SURGICAL TREATMENT OF THE AURICULOTEMPORAL SYNDROME

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ST. LOUIS

THE DISAGREEABLE symptom complex of gustatory sweating was first reported as the auriculotemporal syndrome by Frey.[1] A number of cases have been subsequently reported. Freedberg, Shaw and McManus [2] have reviewed the literature and presented a clinical and pharmacological study of three cases. The condition most frequently follows operations on or injuries to the parotid gland and is characterized by flushing and profuse sweating, upon eating, in the cutaneous distribution of the auriculotemporal nerve. Gustatory sweating has been reported in areas supplied by other divisions of the sensory supply of the head and neck. The frontal area supplied by the supraorbital nerve was involved in the case reported by Tarlov and Herz.[3] Uprus, Gaylor, and Carmichael [4] observed the syndrome in the neck after an operation in the region of the nevus cutaneous colli. Haxton [5] observed involvement of the same area following a cervical thoracic sympathectomy performed through the posterior approach.

The mechanism of this syndrome has been the subject of considerable speculation. Gustatory sweating in the distribution of the sensory nerves of the head and neck is not inhibited by procaine blockade of the cervical sympathetic trunk or ganglia. Sympathetic blocking agents also are ineffective. Freedberg, Shaw, and McManus [2] have postulated that the syndrome is a manifestation of reflexes in which the efferent arc is through cranial sudomotor fibers. They assume nonspecificity of the afferent portion of the arc but suggest that "relief may be obtained by interruption of the efferent arc by alcohol injection or surgical section of the auriculotemporal nerve." Pfeffer and Gellis [6] have reported on a child with bilateral auricolotemporal syndrome developing after drainage of parotid abscesses during infancy. Unilateral procaine block of the auriculotemporal nerve resulted in temporary alleviation of the symptoms on that side. The patient of Tarlov and Herz [3] was cured of frontal gustatory hyperhidrosis by section of the supraorbital nerve.

OPERATION

Two patients with the auriculotemporal syndrome have been successfully treated surgically. The operation was performed under local infiltration. A short incision was made in front of the auricle and auriculotemporal nerve, and superficial temporal vessels were isolated at the level of the temporomandibular joint. The parotid gland was then freed from the cartilaginous external auditory canal.

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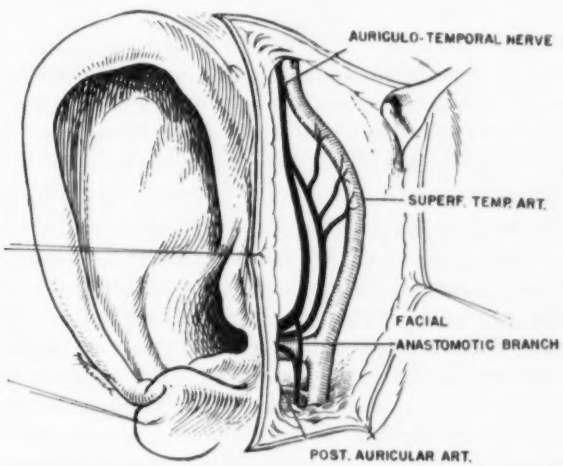
The nerve and the vessels were then dissected proximalward until the facial anastomotic branches were identified. This level was found to be 6 to 8 mm. deep to the posterior border of the ramus of the mandible. Nerve, artery, and vein were then divided proximal to the entrance of the anastomotic branches, and a segment of each was removed. Distal section of these structures was performed just above the zygoma.

REPORT OF CASES

CASE 1 (E. G. Z. 112785).—This 57-year-old, white man was subjected to a radical neck dissection with hemimandibulectomy for carcinoma with bone involvement on March 15, 1949. The primary lesion was in the lip, and bony involvement was secondary to metastases into the submaxillary region. Healing of the wound and recovery were without incident. He was seen at intervals in the Tumor Clinic, and on a return visit in January, 1951, he complained bitterly of excessive sweating involving the right side of the face; this was particularly severe when sour foods were taken. This condition had existed since a short time after his primary operation.



Artist's sketch showing the anatomy of the auriculotemporal nerve and facial anastomotic branches in Case 2. Note three fine filaments blending into the adventitia of the auriculotemporal artery. The superficial temporal vein has been omitted from the sketch. The resection of these structures included all elements visualized in the sketch.

He also complained of pain in the region of the right temporomandibular joint when he talked or chewed, although the mandible had been disarticulated. Procaine blocks of the cervical sympathetic trunk, the lingual nerve, the chorda tympani, and the submaxillary ganglion, and attempted blocks of the sphenopalatine and otic ganglia, as well as methantheline bromide (Banthine) and atropine, failed to alleviate the sweating. Procaine block of the auriculotemporal nerve temporarily eliminated the gustatory sweating. Further investigation was not made. His disability persisted, and on further examination, Aug. 12, 1953, auriculotemporal nerve block was again performed. This block resulted in freedom from the distressing symptoms for a period of three days. A resection of the auriculotemporal nerve and the superficial temporal artery and vein, with identification and section of the anastomosis of the facial to the auriculotemporal, were performed on Sept. 22, 1953. The gustatory sweating was completely eliminated except for a small area supplied by the zygomaticofacial branch of the trigeminal nerve. Resection of these structures resulted also in elimination of the pain which occurred in the temporomandibular joint area when the patient talked or chewed. The distressing symptoms have not recurred.

CASE 2 (V. K. K. 115930).—This 29-year-old, white man had surgical treatment for a mixed tumor of the parotid gland in July, 1952. Approximately one or two days after the operation he began to sweat profusely from the temporal and facial areas. This condition persisted without change until his admission to the Veterans Administration Hospital, Jefferson Barracks, Mo., Jan. 4, 1954. This sweating was produced by the ingestion of any type of food, but was more severe when sour foods were taken. Temperature measurements on both sides of the face revealed that the pathological area was warmer by 1 to 1.5 degrees F. Procaine block of the auriculotemporal nerve gave temporary relief of the gustatory sweating. The nerve and its anastomotic connections with the facial, together with the superficial temporal artery and vein, were resected with local anesthesia, Jan. 8, 1954 (Figure). The wound healed without incident. The distressing symptoms were only partially relieved. Gustatory sweating persists in the distribution of the zygomaticofacial nerve and in the cutaneous distribution of the greater auricular nerve. Postoperatively, a procaine block of the greater auricular nerve eliminated the sweating along the border of the mandible. The volume of the sweating has been materially reduced. The remaining gustatory sweating is hardly noticeable and does not require frequent mopping of the affected side of the face, which was necessary before the operation.

COMMENT

Examination of standard textbooks of anatomy and neuroanatomy shows that cutaneous sensory nerves of the face and neck receive anastomotic branches from the facial nerve. These are reported as joining the various branches of the trigeminal in all its divisions and in particular the auriculotemporal nerve. Communications also exist with the auricular branch of the vagus, the greater auricular nerve, the greater and lesser occipital nerves, and the cutaneous cervical nerves.

It is of particular interest to note that anastomotic filaments from the facial join the lachrymal nerve and the zygomaticofacial branch of the maxillary nerve. In the first case (E. G. Z.) section of the auriculotemporal and the anastomotic facial connections resulted in complete relief from gustatory sweating in the area of cutaneous distribution of the auriculotemporal, but it persisted in the cutaneous distribution of the zygomaticofacial. Hence it is postulated that parasympathetic sudomotor fibers of cranial origin in the efferent arc produce this syndrome and that their resection constitutes an operation which is comparable to sympathectomy for hyperhidrosis in the extremities. The anatomical connections of the facial nerve to the cutaneous nerves of the head and neck are extensive enough to encompass the areas involved in previously reported cases.

There are no reports of this syndrome occurring after radical excision of the parotid gland with sacrifice of the facial nerve. Current techniques in surgery of the parotid for benign conditions involve a simultaneous dissection and preservation of the facial nerve. The region of origin of the anastomotic branches to the auriculotemporal will thus be exposed, and it is suggested that these branches be sought for and sectioned. The approach to this dissection described by Byars [7] will make this extension of the operation relatively simple. The coincident section of the auriculotemporal nerve has previously been advised by Riessner [8] but with another intent, viz., to eliminate secretory fibers and obviate the occurrence of a salivary fistula. I have not had the courage to limit the operation for the auriculotemporal syndrome to division of the anastomotic branches; but the evidence indicates that this syndrome could be cured without producing a sensory deficit, insignificant though it may be.

CONCLUSIONS

Resection of the auriculotemporal nerve and its anastomotic branches from the facial nerve has produced marked relief from gustatory sweating in two patients.

It is postulated that the efferent arc in this syndrome is composed of cranial sudomotor fibers passing by way of the facial nerve to the cutaneous nerves of the head and neck.

The occurrence of this syndrome following operations on the parotid gland may be prevented by the section of the auriculotemporal nerve or the facial anastomotic fibers at the time of the primary operation.

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STUDIES OF THE AGED

Surgical Mortality

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IN A GENERAL hospital serving a rural community, we are often confronted with the problem of major surgery in aged persons. In 1951, at the Mary Imogene Bassett Hospital, 10% of the major operations performed were on patients 80 years of age or over.* One of us (being a surgeon) must often disrupt the physiologic processes of persons in the higher decades of life and the other (an internist) is frequently asked to evaluate and help to repair the disruption in these aged patients. Many times we seemed unable to predict how much surgical trauma could be withstood by such patients and how their convalescence would be affected by the changes of senescence. Although there are many published studies on this subject,† none answered all our questions.

METHOD OF STUDY

The hospital records of all patients more than 69 years of age undergoing major surgery in 1952 and 1953 were reviewed and the following data tabulated: age, sex, type of operation, presence or absence of cardiac disease, overt manifestations of cardiac disease, presence or absence of chronic pulmonary disease, occurrence of complications, and cause of death. Patients who suffered accidental trauma without surgical intervention have been included in those instances in which it was believed that the degree of tissue damage and internal blood loss were equivalent to a major operation.

The patients were divided into two groups, those who survived and those who died after operation. The characteristics of these two groups were compared as binomials and the significance of their differences tested by the chi-square method in the case of enumeration data and by "Student's" t test for mensuration Comparison.

These attributes were compared in an attempt to answer the following questions:

1. In general, what can be learned from the study of the clinical records of these patients that might influence our future handling of surgical problems in the aged?

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From the Department of Surgery and the Cardio-Pulmonary Laboratory, The Mary Imogene Bassett Hospital.

* Powers, J. H.: Unpublished data.

† References 1 to 10.

2. Does increasing age above 70 influence the mortality?

3. Does the presence of cardiac disease influence the rate of fatality?

4. Is any particular type of cardiac disease more significant prognostically than any other type?

5. What is the influence of chronic pulmonary disease on operative mortality?

6. Is there a critical level of cardiopulmonary function which is of prognostic significance?

TABLE 1.—*Operations Survived by Elderly Patients*

	Patients	
	No.	%
Internal fixation of fractured femurs	32	15
Suprapubic prostatectomies	28	13
Hernioplasties	28	13
Operations on the extrahepatic biliary tract	21	8
Colon resections (including abdominoperineal)	20	6
Gastrectomies (including 2 total gastrectomies)	11	5
Suprapubic cystostomies	11	5
Plastic operations on face	9	4
Amputations of lower extremity	7	3
Miscellaneous	48	28
Total	217	

TABLE 2.—*Operations Which Proved Fatal to Elderly Patients*

	Patients	
	No.	%
Internal fixation of fractured femurs	6	33
Cecostomies	3	16.5
Suprapubic cystostomies	2	11
Suprapubic prostatectomy	1	5.5
Radical neck dissection	1	5.5
Laparotomy and biopsy	1	5.5
Ileostomy and subtotal colectomy	1	5.5
Abdominoperineal resection of rectum	1	5.5
Excision of carcinoma of nose	1	5.5
Ventral hernioplasty and resection of gangrenous bowel	1	5.5
Total	18	

7. What complications are prone to develop in the aged, and what is their prognostic significance?

8. What are the causes of death?

RESULTS

From Jan. 1, 1952 to Dec. 31, 1953, there were 240 patients over 69 years of age who fulfilled the requirements of this study. Of these patients, 20, or 8.3%, died. During this two-year period, 1,073 major operations were performed on patients less than 70 years of age. Of these, 21 died, a mortality of 1.9%. The operations performed on the group of patients over 70 that survived are shown in Table 1.

The operations which led to a fatal outcome are listed in Table 2. Two patients who died suffered accidental trauma and did not come to operation.

The mean age of the elderly patients who survived was 76.6 years and that of those who perished, 78.7 years. This difference is not significant.‡

The distribution of sexes did not influence the mortality rate; 56.8% of those who survived and 50% of those who died were men.

The incidence of preexisting chronic pulmonary disease in the two groups was compared. Emphysema and fibrosis were the only pulmonary diseases that occurred frequently enough to be of interest. The diagnosis of fibrosis or emphysema was made only on roentgenologic evidence; so-called senile emphysema was not included unless the radiologist described flattened diaphragms and long, bright, lung fields. An x-ray of the chest was made on 92% of the patients under discussion. One case of pulmonary tuberculosis was discovered, and four patients had asthma uncomplicated by emphysema. None of these died.

TABLE 3.—*Manifestations of Heart Disease in Ninety-Eight Patients*

	Living Group (98)		Dead Group (20)			Statistically Significant
	No.	%	No.	%	△ %	
Congestive failure	23	23.5	4	31	+ 7.5	No
History of congestive failure	22	22	3	23	+ 1	No
Angina pectoris	7	7	3	23	+16	Yes
Hypertension	75	76	11	85	+ 9	No
Cardiomegaly	80	82	10	77	− 5	No
Abnormal electrocardiogram						
Spatial QRS-T angle greater than 50°	55	56	9	69	13	No
Right bundle-branch block	5	5.5	0	0	− 5.5	No
Left bundle-branch block	8		0			
Auricular fibrillation	8	8	3	15	+ 7	No
Miscellaneous changes	6	6.2	3	15	+ 8.8	No

Of the patients who survived, 36, or 16%, had chronic pulmonary disease; this diagnosis was made for 6, or 28%, of those who died ($\chi^2 = 1.74$). We were thus unable to demonstrate a significant influence of chronic pulmonary disease on operative mortality.

Cardiac disease was included only when it produced functional impairment, i. e., congestive failure or a history of failure, angina pectoris, and cardiac enlargement or a major electrocardiographic abnormality. Since the normal limits of spatial QRS-T angle have not been well established in the aged, a wide angle alone was not included as an abnormality. Electrocardiograms were made in 74% of the total group.

Of the entire series, 110 persons, or 45.6%, had objective evidence of cardiac disease. The incidence of arteriosclerotic heart disease with or without hypertension was not significantly different in the two groups. It was the only type of cardiac disease diagnosed, occurring in 57.1% of the patients who died and in 44.5% of those who lived.

‡ The arbitrary age limit of 70 years used in sampling precludes an expression of standard deviation of these means, since the ages are not normally distributed. "Student's" t test of significance, however, is applicable.[11]

Of the various overt manifestations of cardiac disease, only the history of angina pectoris may have been of prognostic importance. Such a history was obtained in only 7% of the group that survived but in 23% of those who died. The chi-square value for this difference is 3.1, a figure which approaches confidence in this difference at the 5% level. We await with interest the accumulation of more cases in order to extend the evaluation of angina pectoris as a hazard to operation. The incidence of the other manifestations of cardiac disease is listed in Table 3.

COMPLICATIONS

The important postoperative complications in these elderly persons concern the heart and lungs. The most frequent are atelectasis and pneumonia, which we have listed together. The importance of these complications is shown by the high incidence of their occurrence in the fatal cases and the significant difference between this incidence of 38% as compared with 14% in the surviving group ($\chi^2 = 6.8$). We have not included the small patches of atelectasis demonstrable at autopsy or by x-ray study which could not be detected by the clinician.

Complications directly attributable to cardiac disease occurred only once in the surviving group, but resulted in death five times, or in 25% of those who died.

The only other important complication that occurred with significant frequency was spontaneous venous thrombosis in 12, or 5.4%, of the patients who lived; 5 of them survived a pulmonary infarct. Three, or 15%, of the group who died had phlebothrombosis and two of those may have had pulmonary emboli.

CAUSE OF DEATH

Consideration of the immediate cause of death indicated that the most frequent single cause was cardiac failure. This occurred six times. On one occasion the patient failed to survive an episode of pulmonary edema secondary to heart failure. Another died of congestive failure and electrolytic imbalance after cecostomy. In three cases myocardial infarction proved fatal. One patient died during an attack of supraventricular tachycardia.

The second most frequent cause of death was atelectasis and pneumonia. This occurred four times in the following ways:

1. F. G., a 73-year-old woman with severe Parkinson's disease, had open reduction and internal fixation of an intertrochanteric fracture of the femur. She died 29 days later. The cause of death as determined by autopsy was diffuse bronchopneumonia. It was believed that the patient's relative immobility and difficulty in swallowing contributed to the development of the pneumonia.

2. J. G., a 77-year-old man, was in an accident in which the wheels of an automobile passed across his chest, producing segmental fractures of two ribs on the right and of six ribs on the left. Tension pneumothorax on the left was successfully treated by tube thoracotomy. Seven days after injury he died with signs consistent with anoxia. Autopsy showed severe chronic lung disease, consisting of fibrosis, emphysema, and bronchiectasis with superimposed atelectasis of the left lung, and a small right hemothorax. It was believed that his pulmonary reserve was not sufficient to sustain life in the presence of the respiratory limitation imposed by his injury.

3. N. D. was 84 years old when she was admitted to the hospital with signs and symptoms of intestinal obstruction secondary to carcinoma of the sigmoid colon. Cecostomy was done. After operation the patient failed to regain consciousness, and five days postoperatively she vomited and aspirated some of the stomach contents. Thereafter there were clinical and x-ray signs of progressive pulmonary consolidation, and on the seventh day she died of pulmonary insufficiency and prolonged hypoxia. Autopsy was not permitted.

4. A. M., an immensely obese 74-year-old woman, had strangulation of bowel in a huge ventral hernia which had been unreducible for five years and symptomatic for three days. Three hundred and fifty centimeters of gangrenous small bowel was resected and the hernia repaired. For five days thereafter she did well. On the sixth day, cyanosis accompanied the development of physical and x-ray signs of atelectasis. In spite of treatment aimed at relieving the atelectasis, evidence of anoxia and infection progressed, and she died of pulmonary insufficiency eight days after operation. Autopsy showed atelectasis sufficient to explain her death.

Three patients died suddenly and unexpectedly of undetermined cause. In two instances it was believed that death was due to pulmonary embolus.

The remaining six patients died of the variety of causes listed below.

(*a*) Volvulus and gangrene of cecum after resection of rectum

(*b*) Retroperitoneal hemorrhage from a sclerotic blood vessel

(*c*) Acute and chronic intestinal obstruction and chronic peritonitis secondary to chronic ulcerative colitis and ileitis

(*d*) Neurogenic hypernatremia secondary to papilloma of choroid plexus

(*e*) Proteus septicemia resulting from urinary tract infection

(*f*) Hemorrhage from urinary tract after prostatectomy and incidental perforation of intra-abdominal viscus (presumably a peptic ulcer)

In six of the patients who died (30%) the immediate cause of death was either preventable or reversible. In retrospect, each of these six deaths might have been forestalled by better judgment or more accurate technique on the part of the clinician.

1. F. D. was 77 years old when he was admitted to the hospital because of urethral obstruction secondary to benign hypertrophy of the prostate. He had a history of angina pectoris and signs of arteriosclerotic heart disease. A transurethral resection of the prostate was performed, during which extraperitoneal perforation of the bladder occurred. A few hours later suprapubic cystostomy was done because of this operative complication. His course was then one of progressive deterioration with heart failure. A diagnosis of acute myocardial infarction, without pain, antedating his operation by a da yor two had been made by electrocardiogram, but this information had not reached the surgeons before operation.

It is apparent that had the diagnosis of myocardial infarction been known the patient would not have been subjected to operation and might have survived the coronary occlusion of which he died.

2. M. V. H. was 86 years old when she fell and sustained an intracapsular fracture of the neck of the left femur. She was admitted to the hospital a few hours thereafter and was found to be in apparently good physical condition except for unexplained chronic anemia; her blood contained 8.5 gm. of hemoglobin per 100 cc. Consequently, in the course of the next 12 hours, she received 1,500 cc. of whole blood intravenously and was then subjected to removal of the femoral head and

replacement with a Judet prosthesis. Immediately after operation she developed signs of pulmonary edema and a concomitant fall in arterial blood pressure. The condition did not respond to treatment, and she died eight hours later.

The effect of large-volume transfusion in patients with chronic anemia has been studied [12] and its dangers pointed out. Its adverse effects under these circumstances result from a fall in cardiac output secondary to an increase in right atrial pressure.

In retrospect, this patient should have been treated in traction for a few days until the anemia had been corrected more slowly.

3. P. V. D. was 82 years of age. She had a long history of arterial hypertension but was active and apparently well until she fell from a stepladder and sustained a fracture of the neck of the left femur. At the time of admission to the hospital, her blood pressure was 220/150 and she had signs of minimal congestive failure. She was digitalized, and about 24 hours after injury the fracture was reduced and fixed with a Smith-Petersen nail.

During the operation a fall in arterial blood pressure occurred. This was treated as if it resulted from blood loss. Immediately after operation, signs of pulmonary edema were observed but incorrectly interpreted. She died 18 hours after operation, having been treated by blood transfusion, placed in the Trendelenburg position, and given oxygen.

This patient had unrecognized pulmonary edema, and here again treatment was misdirected.

4. F. M. was 71 years old when he underwent abdominoperineal resection of the rectum for carcinoma. Two days thereafter he began to complain of pain in the right lower abdominal quadrant. Signs of inflammation appeared in that area, and he was thought to be developing an intraperitoneal abscess. Five days later his abdomen was reexplored, and volvulus of the cecum and gangrene were found. He subsequently developed subphrenic abscess and empyema and died of overwhelming sepsis.

If the diagnosis of gangrenous bowel had been made earlier, he probably would have been saved by resection.

5. C. S., a 70-year-old man with chronic pulmonary disease, underwent excision of a carcinoma of the nasal vestibule and closure of the wound by means of a flap rotated from the cheek. On the second day after operation he complained of substernal pain radiating into his arms, accompanied by a drop in arterial pressure. A diagnosis of myocardial infarction was made. He died five hours after onset of symptoms. Autopsy showed his heart to be enlarged but that the coronary arteries were widely patent. No recent myocardial infarct could be demonstrated.

When the electrocardiograms were reviewed, it was evident that the changes shown were not due to myocardial infarction but rather to supraventricular tachycardia with a rate of 160 beats a minute. It is likely that had the nature of the arrhythmia been recognized and steps taken to convert it to sinus rhythm his death might have been averted.

6. A. S., an 86-year-old woman, was admitted to the hospital with symptoms and signs of obstruction of the left side of the colon. Sigmoidoscopy revealed an annular mass occluding the lumen at 12 cm. Biopsy of the tissue was done, and cecostomy was performed with the patient under local anesthesia. After operation she became disoriented and lethargic; ventricular fibrillation and massive peripheral

edema developed. The serum sodium concentration fell to a low level (120 mEq. per liter) in spite of the infusion of salt solution. She gradually became more lethargic and died six days after cecostomy.

Autopsy showed chronic passive congestion of lungs and liver, together with peripheral edema and small hydrothorax bilaterally. No lesion of the intestinal tract was demonstrated.

In restrospect, it appears that this patient's symptoms may have been due to chronic passive congestion. The sigmoidoscopic findings had obviously been misinterpreted. Had cecostomy been avoided and treatment concentrated on her cardiac disease, she might have recovered.

Study of this group of patients, whose deaths were due to errors in interpretation or judgment, points to the great importance of accurate diagnosis in surgical treatment of the aged. In each instance the diagnosis of the lethal complication was incorrect. In two cases pulmonary edema was precipitated by overenthusiastic fluid replacement. In one instance the presence of a painless myocardial infarction was not recognized before operation, while in another a potentially reversible arrhythmia was misdiagnosed as myocardial infarction. In the two remaining cases there was a gross error in the diagnosis of an intra-abdominal condition, leading to unnecessary operation in one case and the withholding of a necessary one in the other.

As far as we know, there are no published studies of operative mortality in the aged in which fatal cases have been analyzed from the standpoint of the avertability of death. Our over-all mortality rate of 8.3% in this age group is lower than that of most reported series.§ Yet if the avertable deaths be deleted, the mortality falls to 5.8%. Even this figure is not directly comparable with the fatality rate of 1.9% at this hospital among patients below the age of 70. The statistic is confounded by the fact that many less serious operations and more elective procedures are performed in the younger group. Our available figures do not allow a quantitative estimate of the influence of this factor on the difference.

No component of the difference in fatality rate between our elderly patients and those below 70 has been shown to be dependent upon old age itself. Rather, our data suggest that the difference results from lack of tolerance of the elderly to postoperative complications of the cardiorespiratory tract and to diminished tolerance to misdirected therapy.

We find this concept stimulating, first, by serving to dispel the aura of pessimism that so often surrounds the elderly patient with a surgically correctible condition and, second, by stimulating our interest in efforts to improve the surgical results in this age group.

SUMMARY AND CONCLUSIONS

In the years 1952 and 1953, there were 240 patients over 69 years of age who had major surgical operations at the Mary Imogene Bassett Hospital. Their mortality rate was 8.3%, as compared with an operative mortality of 1.9% in patients less than 70 years of age. The reasons for this significant difference are discussed.

We have been unable to show that after the age of 70 further increase in age has any bearing on the mortality rate.

Arteriosclerotic cardiac disease existed with equal frequency in those who survived operation and in those who died. Of the various manifestations of cardiac

§ References 1 to 9.

disease, only the existence of angina pectoris may have been associated with a poorer prognosis. Although preexisting congestive failure could not be shown to alter the prognosis, its development in the postoperative period was associated with a distinctly higher mortality rate.

Chronic pulmonary disease was as common in the patients who survived operation as in those who died. Postoperative pulmonary complications in the form of atelectasis and pneumonia were, however, significantly more frequent in the patients who died.

The commonest causes of death were postoperative complications of the cardiopulmonary system.

We were not able to determine a critical level of preoperative cardiopulmonary function which was of prognostic significance.

Thirty per cent of the deaths may have been preventable. The significance of this high incidence of diagnostic and therapeutic error is discussed.

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LYMPHOMAS OF SPINAL EPIDURAL SPACE

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INVASION of the spinal epidural space by lymphoblastic tissue is an infrequent complication of Hodgkin's disease or lymphosarcomatosis. Signs and symptoms of involvement of the central nervous system in lymphoblastic disease are not unusual but actual invasion of the brain and spinal cord is extremely rare. Symptoms referable to the spinal cord in patients who have these lesions most often arise as the result of pressure on the cord occurring after collapse of an involved vertebra.

In most patients the primary lymphoblastomatous lesions originate elsewhere and secondarily involve the central nervous system. We have seen a number of cases in which there appeared to be primary involvement of the spinal epidural space without evidence of tumor elsewhere. These cases form the basis of this report.

MATERIAL

Search of the records at the Mayo Clinic revealed 39 cases in which the primary symptoms of tumor of the spinal cord were due to lymphoblastomatous lesions in the absence of clinical evidence of involvement elsewhere in the body.

The records in these 39 cases have been reviewed as to age, sex, symptoms, roentgenologic evidence of tumor, surgical findings, and pathologic diagnosis. A postoperative follow-up has been attempted. Twenty-three of these cases were included by Kloos in his thesis presented in 1943 to the Graduate School of the University of Minnesota. Pathologically the lesions were classified similarly to those of Kloos. The categories included Hodgkin's disease, reticulum cell sarcoma, lymphosarcoma (small cell type), follicular lymphoblastoma, and a mixed type of tumor.

SYMPTOMS AND SIGNS

Of these 39 patients who had lymphoblastic lesions involving the spinal cord, 27 (69%) were male and 12 (31%) were female. All age groups were represented (Table 1); the youngest in the series was an 8-year-old boy who had reticulum cell sarcoma, whereas the eldest was a 79-year-old man who had Hodgkin's disease. However, the tumors occurred more commonly in patients in the older age groups. Systemic lymphoblastoma occurs more often in younger patients.

The Mayo Foundation is part of the Graduate School of the University of Minnesota.

From the Section of Neurologic Surgery (Dr. Love); Assistant to the Staff (Dr. Miller), and Section of Pathologic Anatomy (Dr. Kernohan), Mayo Clinic and Mayo Foundation.

The distribution of these tumors in the spinal canal corresponded to the prevalent sites of tumors of the spinal cord reported by Rasmussen and associates, the thoracic level being involved most commonly (25 cases, 64%); the lumbar or lumbosacral region was involved in 13 cases (33%) and the cervical level in 1 case (3%).

Pain and weakness of the extremities were the most prominent presenting complaints. The duration of symptoms varied from two days to one year. Commonly, four to five months elapsed between the onset of symptoms and registration at the clinic.

TABLE 1.—*Age Incidence of Lymphoma of the Spinal Epidural Space*

Age, Years	Cases
1-10	2
11-20	4
21-30	4
31-40	2
41-50	9
51-60	8
61-70	9
71-80	1
	39

TABLE 2.—*Symptoms and Signs in 39 Cases of Lymphoma of the Spinal Epidural Space*

Symptom	Cases	Per Cent
Pain	33	85
Night pain	5	13
Weakness	30	77
Paresthesia	24	62
Incontinence	20	51
Sign		
Sensory change	30	77
Weakness	28	72
Reflex changes	30	77
Paraplegia	7	18
Subarachnoid block	26	67
Increased total protein	28	72
45-100 mg.	7	
101-500 mg.	6	
501-1,000 mg.	2	
1,001-4,000 mg.	6	
Fluid yellow; coagulated on standing	7	

The most frequent symptoms referable to the central nervous system were pain, weakness, and paresthesia (Table 2). Twenty patients had incontinence of urine and feces.

Sensory deficits were present in 30 of the 39 patients (77%) and weakness of the extremities without paraplegia was noted in 28 (72%); in 7 patients (18%), complete paraplegia with a sensory level was present. One patient who had a thoracic lesion presented a classic Brown-Séquard syndrome that disappeared after surgical removal of the tumor. Changes in reflexes were present in 30 of the 39 patients (77%). Tumors above the level of the conus medullaris were prone to cause an increase in the deep tendon reflexes, while tumors below this level produced diminution of the reflexes.

Plain roentgenograms of the spinal column revealed evidence of an intraspinal lesion in 13 of 39 cases (33%), the commonest disturbance being erosion of the

body of the vertebra and of the pedicles of the vertebral body (Fig. 1 *a*). Erosion of the bone surrounding the intervertebral foramina was present in several cases (Fig. 1 *b*). However, none of these findings were attributed to lymphoblastic lesions preoperatively. Myelography, using ethyl iodophenylundecylate (Pantopaque), revealed evidence of an extradural tumor in all eight patients in whom it was done (Fig. 2).

Spinal punctures were performed preoperatively in 28 patients. Evidence of a block in the subarachnoid pathways was present in 26 of these 28 patients (93%); an increase in the protein in the cerebrospinal fluid was present in all 28 patients,

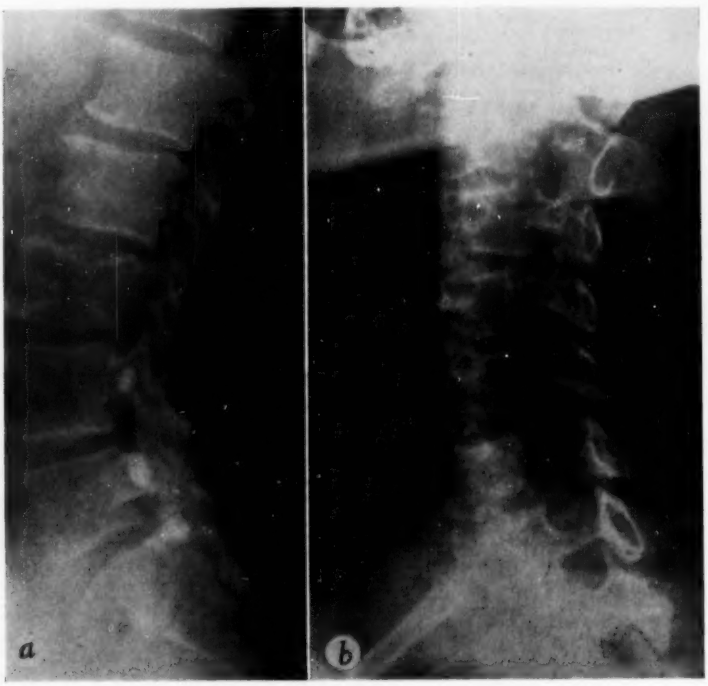

Fig. 1—(*a*) Lateral roentgenogram of the lumbar portion of the spinal column of a 57-year-old man. The body of the third lumbar vertebra is narrowed (pathologic compression fracture), with decreased density owing to invasion of the vertebral body by lymphoblastoma. Questionable involvement is present in the body of the fourth lumbar vertebra. (*b*) Left oblique roentgenogram of the cervical portion of the spinal column of a 47-year-old man. Lymphosarcoma has extruded through and eroded the edges of the left fourth, fifth, sixth, and seventh cervical intervertebral foramina. There is also erosion of the bodies of the cervical vertebrae.

the values ranging from 45 to 4,000 mg. per 100 cc. (Table 2). No abnormal increase in the number of cells in the fluid was observed. The erythrocytic sedimentation rate was increased in only eight patients.

PATHOLOGY

The classification of neoplasms of lymphatic tissue is a controversial subject. The term "lymphoblastoma" is widely used as a group designation for tumors originating in lymphatic tissue. Mallory, in 1914, used the term first in referring to

tumors derived from the lymphoblast. Exception to the use of lymphoblastoma as an over-all classification was taken by Krumbhaar and Watson because they considered that it does not include Hodgkin's disease.

Subclassification may be based on histopathologic changes, histogenesis, clinical symptoms, location of lesions or combinations of these factors. Some authors include leukemia in the classification, but since leukemic lesions of the spinal cord are usually multiple and widespread, offering ample evidence of lesions elsewhere, this condition is not included in this study.

Fig. 2—Myelogram revealing a large extradural reticulum cell sarcoma in a 65-year-old man. The tumor extended from the sixth cervical to the second thoracic vertebra as verified at operation. The plain roentgenograms of the cervical region in this patient showed no abnormalities.

We have chosen the group designation of "lymphoblastoma," with two subdivisions, namely Hodgkin's type and lymphosarcoma. When possible, the latter is divided into small cell type, reticulum cell sarcoma, and follicular lymphoblastoma. No instances of the latter lesion occurred in our series.

Microscopically, Hodgkin's disease is described as a lesion containing a reticulum in which lie a few small lymphocytes, large lymphocytes, plasma cells, eosinophiles, proliferating endothelium and endothelial giant cells (Fig. 3*a*). Ewing stated that there are many variations in the proportions of these elements. Most pathologists agree with this description. The cause of Hodgkin's disease is debated. Some observers, including Karsner, Ewing, and Reed, considered that this lesion

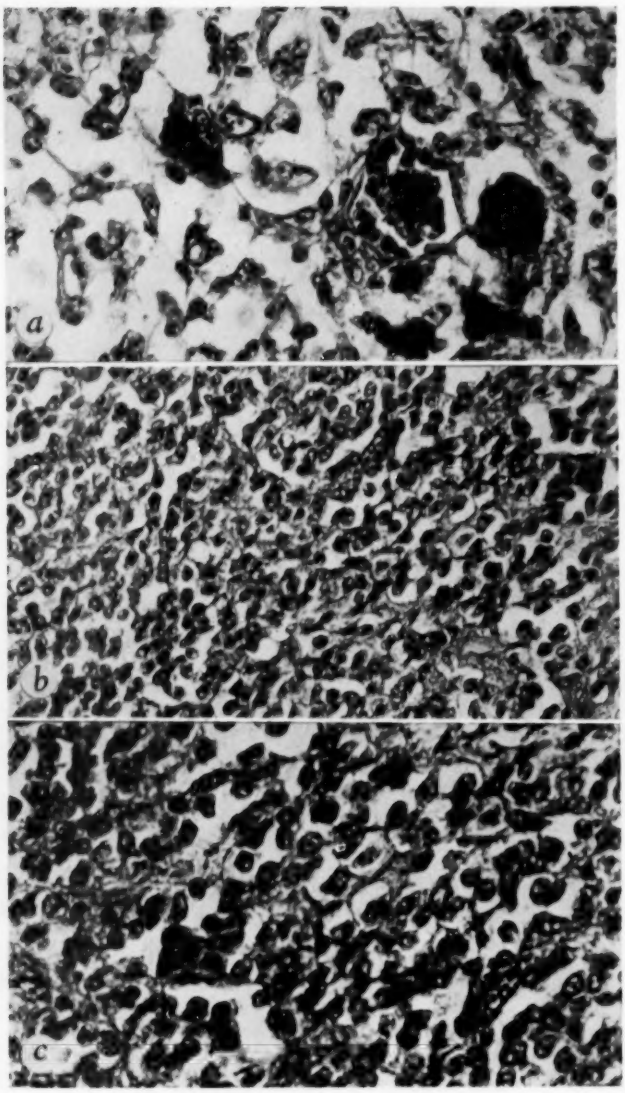

Fig. 3.—(*a*) Lymphoblastoma, Hodgkin's type, from a 24-year-old woman (eleventh thoracic vertebra). Note large multinucleated cells, mature lymphocytes and reticulum cells. (*b*) Lymphosarcoma, small round cell type, from a 49-year-old man (eighth thoracic vertebra). Note numerous small dark-stained nuclei. A few larger reticulum cells are present. This is somewhat similar to *a* but the cells are much smaller and the nuclei are darker and more numerous. (*c*) Reticulum cell sarcoma from a 24-year-old woman (upper thoracic extradural space). The nuclei and cells are larger than those seen in *b* and a fine reticular network is in the background (all hematoxylin and eosin; ×500).

is of granulomatous origin. Warthin and Weber thought that the disease is definitely a neoplasm and that reticulocytosarcoma and lymphosarcoma may be transitional forms of atypical Hodgkin's disease.

Lymphosarcoma is regarded as being a neoplasm by most pathologists. Microscopically, this tumor as it occurred in our series was divided into two types as follows: (1) small cell lymphosarcoma, in which the cells correspond to the size of lymphocytes (Fig. 3 *b*), and (2) reticulum cell sarcoma, which is composed of larger cells corresponding to reticuloendothelial cells (Fig. 3 *c*). The cells in both types may be uniform in size and shape. Multinucleated cells may be present in either form and mitotic figures are frequent. Watson was of the opinion that the cellular elements may be lymphocytic, lymphoblastic, or reticular, the lymphoblasts being larger than lymphocytes and showing a clear nucleus, often containing a

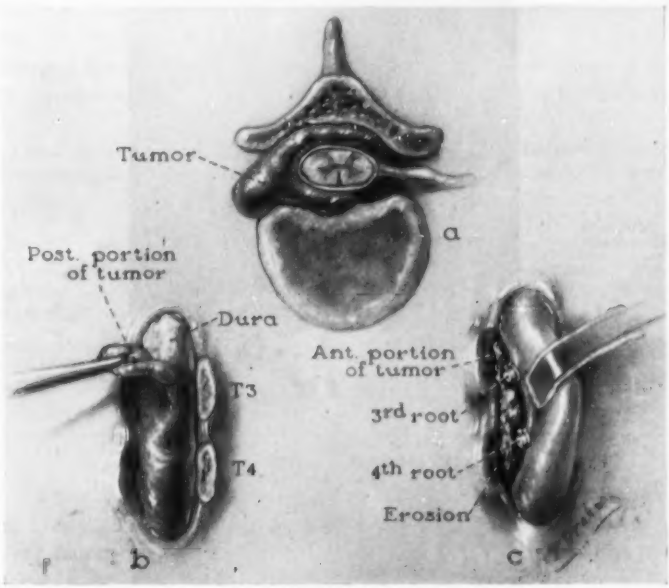

Fig. 4.—Drawings made during operation on a 41-year-old man. Note the extradural lymphoblastoma encircling and compressing the dura and spinal cord and eroding the third and fourth thoracic vertebrae and their laminae. The left third and fourth thoracic nerve roots were divided to facilitate removal of the tumor and to allow the spinal cord to ride more freely on the remaining tumor.

nucleolus, and strongly basophilic cytoplasm. The histologic differentiation of lymphosarcoma frequently may be difficult because the component cells vary in appearance and number. Callender stated that Hodgkin's disease sometimes cannot be differentiated from reticulum cell sarcoma.

Histopathologic study of the lesions in this group of 39 cases revealed that 14 lesions were classed as lymphosarcoma, small cell type, whereas 11 were reticulum cell sarcoma. Hodgkin's disease occurred in seven cases. In six cases, the tumors presented the microscopic picture of a mixture of small and large cell sarcoma; these lesions were classified as a mixed type of tumor. In one case the tumor could not be classified accurately.

TREATMENT AND RESULTS

All 39 patients underwent operation. The extent of the surgical procedure depended on the general condition of the patient. In some, only biopsy was done for verification of the type of tumor present, while in others extensive removal was attempted. If the nerve roots were involved and pain was a prominent symptom, rhizotomy of the involved nerve root was performed (Fig. 4). Thirty-six of the 39 patients received postoperative roentgen-ray therapy in the involved region (Fig. 5). Only one patient in this group received cortisone and no relief was obtained.

Fig. 5.—Postirradiation roentgenogram of the patient mentioned in connection with Figure 1*b* taken 45 days after start of roentgen-ray therapy. Note practically complete regeneration of the edges of the cervical foramina and the vertebral bodies.

Postoperatively, 24 patients (62%) showed improvement in their neurologic deficit, while 15 patients failed to improve. The latter patients were in the older age groups, and it is assumed that irreversible vascular changes had occurred in the spinal cord. Only 11 of the 36 patients (31%) who received irradiation showed improvement as the result of such treatment, whereas 6 (17%) were worse.

FOLLOW-UP STUDY

A review of the records disclosed that 22 patients (56%) were dead and 14 (36%) alive at last report, while 3 (8%) were untraced (Table 3). Patients who had reticulum cell sarcoma appeared to survive longer than did those who had any of the other types of tumor.

COMMENT

It is difficult to determine the origin of lymphoblastoma occurring in the spinal epidural space. The lesions may be metastatic from distant parts of the body or they may be primary in the epidural space. Metastatic lesions may occur after hematogenous spread. As previously indicated, the cases included in this study offered no evidence of a primary lesion elsewhere at the time of examination.

Direct extension into the epidural space from nearby tissue through the intervertebral foramina has been advocated as the source of epidural lymphoma by Drake and by East and Lightwood. Small neoplastic masses in the subpleural, mediastinal or retroperitoneal lymph nodes could easily escape detection. The vertebral body may be a source of the tumor according to Weil and Winkelman and Moore. As already mentioned, 13 of our 39 patients had roentgenologic evidence of changes in the vertebral bodies but in no instance was the lesion thought to be lymphosarcomatous on the basis of this evidence.

Iwanow and Romodanowsky have shown that lymphatic drainage of the spinal canal is to the mediastinal and retroperitoneal lymph nodes. The lesions may spread

TABLE 3.—*Survival in 39 Cases of Lymphoma of the Spinal Epidural Space*

		Alive			Dead			
			Time After Operation			Time After Operation		
Type of Lymphoma	Total	Cases	Months	Years	Cases	Months	Years	Unknown
Reticulum cell sarcoma	11	4	8	8, 9, 10	6	3, 5	1, 1, 2, 8	1
Lymphosarcoma, small cell	14	5	1, 4	2, 3, 4	8	1, 1, 4, 7, 8, 11	5, 6	1
Hodgkin's disease	7	3	6	1, 18	4	4, 8	1, 3	..
Lymphoblastoma, mixed type	6	1	..	1	4	6	1, 2, 5	1
Unclassified	1	1	2	..	..	..	..	..
Total	39	14	..	..	22	..	..	..

by direct extension into the epidural space from along this route. The presence or absence of lymphatic tissue in the spinal canal is not definitely known. Browder and de Veer found no aggregation of lymphatic tissue on examination of the epidural space in a series of persons who died accidentally, but they stated that small foci of lymphatic tissue may be present. Blakeslee, as well as Drake and Elsberg, suggested that small lymphatic elements are present in the epidural fat.

The reticuloendothelial system is coextensive with the entire body; if one considers that lymphosarcomas arise from this tissue, then there is a distinct possibility that these tumors occur primarily in the spinal epidural space.

Among the patients who were relieved after operation, 18 experienced great improvement but returned within 1 to 48 months (average 11 months) because of recurrence of symptoms. Further roentgen-ray therapy was of benefit in these patients.

One patient, aged 53, obtained great relief of symptoms from decompression of the spinal cord and removal of a large mixed lymphoblastoma at the level of the sixth to the eighth thoracic vertebrae. Operation was followed by increase in blood pressure from 150 mm. of mercury systolic and 100 diastolic to 210 mm. of mercury systolic and 150 diastolic; the patient died later as the result of a cerebrovascular

accident. It is suspected that the compression of the cord in this patient resulted in a sympathectomy-like effect on the peripheral circulation and that this beneficial effect disappeared when the cord was decompressed, thus accounting for the subsequent increase in blood pressure. An account of this case follows.

REPORT OF CASE

A 53-year-old white man came to the clinic because of progressive weakness of his legs of 10 months' duration. He had noted progressive pain in the thoracic portion of the spinal column, as well as malaise and chills, for two months. Six weeks prior to his admission he noted inability to void.

Examination at the clinic revealed a sensory deficit up to the seventh thoracic dermatome, moderate weakness of the legs, and absence of the knee and ankle jerks. Results of routine hematologic studies were within normal limits. Roentgenograms of the thorax showed no abnormalities. Roentgenograms of the spinal column revealed compression of the body of the third lumbar vertebra and moderate spondylolisthesis of the lumbosacral joint. Differential spinal punctures disclosed complete block of the subarachnoid pathways at the level of the seventh thoracic vertebra. The cerebrospinal fluid below the block was yellow and contained 2,000 mg. of protein per 100 cc.

Extensive thoracic laminectomy revealed a reddish-gray, vascular, extradural mixed type of lymphoblastoma that extended from the sixth to the ninth thoracic vertebra; it was attached to the extradural nerve roots. The tumor was dissected from the dura, after which the dura pulsated freely.

The patient's convalescence was uneventful. He received roentgen-ray therapy in five divided doses. Gradual improvement was apparent in the sensory deficit and the strength of his legs. Fourteen months after the operation he was able to walk with only a cane for support. Improvement also occurred in vesical function. As already indicated, his blood pressure increased in the ensuing year and he died 16 months after operation as the result of a cerebrovascular lesion.

The ultimate results of treatment of these lesions is not encouraging, as seen in Table 3. Although an occasional patient may survive for a long period, the final outcome usually was decided within a few months. Only one patient in this group was treated with cortisone, and the results were discouraging. This corresponds to the results of treatment of generalized lymphosarcoma with cortisone and corticotropin (ACTH). Stickney reported that use of cortisone will alleviate the acute subjective symptoms of lymphoblastoma but not affect the anatomic changes; he stated that the best over-all therapy is irradiation combined with administration of nitrogen mustard.

As is common with other tumors, the patients afflicted with this disease were primarily in the middle age groups. Males were more prone to have the disease than were females. This agrees with the findings of Minot and Isaacs regarding sex incidence of lymphoblastoma in general. Pain and weakness, common symptoms with tumors of the spinal cord, were the most common complaints presented by patients who have lymphoma of the epidural space. This corresponds to the statements of Craig and of Grant that the outstanding early symptom of extramedullary intraspinal tumors is pain. This symptom is due to involvement of the nerve roots at the level of the tumor. As the tumor enlarges, compression of the cord occurs, with resultant weakness of the muscles below the level of the tumor, paresthesia, and disturbances in bowel and bladder.

In view of the fact that roentgenographic evidence of tumor was present in only 13 of 39 patients, it would appear that a negative roentgenogram has little value in the diagnosis of possible extradural lymphoblastomas. On the other hand, 26

of the 28 patients subjected to spinal puncture had evidence of block of the spinal subarachnoid pathways, and the values for protein in the cerebrospinal fluid were increased in all 28 of these patients.

Laminectomy and decompression of the involved segment of the spinal cord and nerve roots with rhizotomy of the involved roots in some instances brought improvement of the neurologic status in 24 of the 39 patients. As mentioned before, 11 patients showed improvement after roentgen-ray therapy. It would appear that decompression plus irradiation is the treatment of choice in these patients. However, one must be prepared to lose some of the benefit gained by operation in some instances, as six patients were worse after irradiation.

SUMMARY

A study has been made of the records of 39 patients encountered at the Mayo Clinic who had lymphoblastomas of the spinal extradural space without clinical evidence of tumor elsewhere.

Lymphosarcoma of the small cell type was the most prevalent kind of tumor present, followed by reticulum cell sarcoma and Hodgkin's disease in that order. Six cases of lymphoblastoma, mixed type, were present.

Pain, weakness, and paresthesia were the most common presenting subjective symptoms, while sensory defects and muscular weakness were the most common neurologic signs. No definite method was found to make a diagnosis of lymphosarcoma prior to operation. The treatment of choice appeared to be surgical decompression of the involved portion of the spinal cord followed by irradiation therapy. Use of nitrogen mustard might be tried.

Twenty-two of the 39 patients were dead at last report. A period of three months to eight years elapsed from onset of symptoms to death. Fourteen patients were alive 1 month to 18 years after operation.

Evidence exists that lymphoblastomatous lesions can occur primarily in the extradural space.

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BILE PERITONITIS DUE TO SPONTANEOUS PERFORATION OF THE COMMON DUCT

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BILE PERITONITIS due to a spontaneous perforation of the common duct without antecedent history of trauma, previous surgery or obstructive calculi is exceedingly rare. Spontaneous perforation of the common bile duct in this category has been reported at infrequent intervals, possibly due to one cause or to a combination of causes which include (*a*) increased intraductal pressure due to spasm of the sphincter of Oddi; (*b*) intramural infection; (*c*) diverticulum; (*d*) necrosis of the part of the wall due to a mural vessel thrombosis; (*e*) necrosis of the wall secondary to a calculus, and (*f*) reflux pancreatic secretions and tissue digestion.* As early as 1912, McWilliams [1] reviewed 90 cases of bile peritonitis due to spontaneous rupture of the biliary tract of all causes and showed that 91% occurred in the gall bladder, 4.4% in the common duct, 3.3% in the cystic duct, and 1.1% in the hepatic duct. This serves as a general comparison of the incidence of the common duct perforation with that of the rest of the biliary tree.

A case of spontaneous perforation of the common duct of unknown etiology, with recovery, follows.

REPORT OF A CASE

A 65-year-old white man was admitted to the Veterans Administration Hospital, Lyons, N. J., on Dec. 14, 1946, with a diagnosis of schizophrenic reaction, catatonic type. He was supposedly catatonic and mute. On the morning of March 19, 1951, he cried out with pain and pointed to the right upper quadrant of the abdomen. His temperature was 98.6 F., pulse rate 66, and respiration rate 18. No inventory of systems could be obtained, nor could any pertinent information relevant to illnesses, operations, or accidents be elicited. During his five-year institutional stay, no serious illness was recorded. He had an artificial right eye, with no history in reference to etiology. Examination revealed a mild-looking, lean, elderly man, suspicious, uncooperative, resistive to examination, mute, but with apparent abdominal distress. The blood pressure was 128/88. No jaundice was noted. Heart and lungs were normal. The patient pointed to the right of the abdomen above the level of the umbilicus. There was tenderness and spasticity of the entire abdomen, more marked in the upper right quadrant. Although there was no vomiting or diarrhea, it was found that he refused breakfast that morning. On inspection, a globular mass was seen in the hypogastrium, representing a dis-

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* References 2, 10, 12, and 14.

tended bladder. This receded completely after catheterization. Tenderness and spasticity still persisted. Rebound tenderness could not be elicited with satisfaction. No masses were palpable, but most resistance was felt in the right upper quadrant. The rest of the findings were noncontributory.

X-ray examination of the abdomen failed to show any evidence of intestinal obstruction, free intra-abdominal air, or soft-tissue masses. X-ray examination of the chest was negative. Blood serum amylase was 133 units; WBC 15,000, with 86% neutrophiles and 12% lymphocytes, and RBC 4,350,000, with 14 gm. of hemoglobin per 100 cc.

In view of the lack of history on a catatonic patient and since all signs pointed to the upper right quadrant, with an elevated blood count, the differential impression entertained was (1) acute cholecystitis and (2) penetrating peptic ulcer. He was placed under observation, with bed rest, sedation, elimination of oral intake, and administration of intravenous fluids. The temperature rose to 100 F. at night. The following morning the temperature was normal, but the patient vomited and the WBC increased to 20,800, with 87% polymorphonuclears. The abdomen was still tense, with marked tenderness and splinting in the right upper quadrant. Not to risk further complications, exploratory laparotomy was proposed.

After the abdomen was prepared with pHisoderm (a soapless, sudsing detergent cream) and draped with sterile towels to expose the right abdomen, a right upper paramedian incision was made, retracting the rectus muscle laterally. When the peritoneum was opened, about 500 cc. of clear bile was expressed. Thorough exploration of the abdomen, excepting the biliary tract, failed to show any evidence of pathology. The liver was smooth and of normal color, although there were areas exhibiting some fibrosis. The gall bladder was small, thick-walled, and contracted, but failed to show any area of perforation. Exploration of the lesser sac did not reveal bile. After adequate display of the hepatoduodenal ligament, no gross pathologic process was noted nor was there evidence of bile extrusion anywhere. However, a small rent in the peritoneum of the hepatoduodenal ligament was noted on its most lateral aspect, about 1 cm. distal to the entrance of the cystic duct into the common duct. The peritoneum was stripped from the common duct, and a small perforation the size of a pin point was noted on the anterolateral aspect of the common duct. Despite the fact that no stones were felt in the gall bladder, it was felt that it was reasonable to remove the gall bladder because of the possibility of a rent in the gall bladder itself, which might have been overlooked on exploration. The gall bladder was removed in retrograde fashion, and, when the ampulla was found, it was noted that the hepatoduodenal ligament was wanting on the superior aspect of the cystic duct. The common duct was explored; a clear bile was obtained, and there were no stones. A T-tube was inserted and brought out through a stab wound in the right flank, together with a drain to Morison's pouch. Figure-of-eight, steel-wire sutures closed the peritoneum and fascia. Interrupted fine cotton was used for the skin. Transfusion of 500 cc. of blood was given during the procedure. General oxygen-ether anesthesia was administered, and the patient tolerated the procedure well.

Gross and histologic examinations of the gall bladder showed acute and chronic cholecystitis.

The postoperative course was satisfactory, considering that the patient was a catatonic. Atelectasis during the first five days was treated by balance of input and output and administration of penicillin (100,000 units q. 3 h.; the patient was out of bed on the third day). Oral alimentation was poor, but this improved gradually. Cholangiograms were performed during the third and fifth postoperative weeks, which did not demonstrate obstruction. T-tube was removed during the sixth week. Recovery was complete. Follow-up examinations 7 and 18 months later showed a favorable condition.

COMMENT

Perforation of the gall bladder secondary to obstructive cholecystitis or empyema of the gall bladder is common, but perforation of the biliary duct system in the presence of this infection is exceedingly rare. The majority of cases reported are associated with trauma or previous surgery.[2] Gans and Reydaman [2] and others report a case due to erosion of a calculus. When no calculi are apparent, one must determine the etiological factors in perforation of the common duct. Bailey,[3] Vale and Shapiro,[5] and Newell [6] each report one case of a perforation of the choledochus

not associated with trauma, surgery, or impacted stone. Newell [6] found no obstruction at any point in the common duct or the hepatic ducts to account for the rupture. He explained it on the basis of an acute choledochitis combined with spasm of the sphincter of Oddi, resulting in a temporary partial obstruction which in turn produced an empyema of the duct and rupture at its weakest point.

Reich [7] reported a similar case involving the hepatic duct. Smith [4] also described a case of rupture of an intrahepatic duct with escape of bile from the surface of the liver.

Still there have been cases of diverticula of the choledochus with succeeding perforation. Wernsdorfer [13] had one case due to diverticulum, with infection and recovery. Blegen and Boyer [16] demonstrated a case due to diverticulum and stenosis of the common duct, with death. Taube and Himel [11] showed one due to cyst of the common duct, with recovery.

Burden [8] mentioned an etiologic doctrine and invited the possibility of a small stone arising in the glands of the bile duct, imbedded in the wall of the inflamed duct producing a rupture. He also pointed out that the biliary ducts contain numerous glands in their walls extending almost to the peritoneal coat. Normally, these sacculi contain no bile but contain the mucous secretions of these glands. In a dilated and diseased duct these glands may be the seat of infection and calculus formation; under cholangitis, with increased intraductal pressure, perforation may result.

It might be noted here that Walters and Snell [9] remark that the mucosa of the common duct is changed very little in the majority of cases of stones in the common duct. They declare this natural resistance of the epithelial surface to be remarkably high to trauma.

Hart [12] recently described a case due to gangrene of the common duct with perforation, and he thought the local gangrene due to thrombosis of mural vessels. These vessels probably are derived from branches of posterior-superior pancreaticoduodenal artery.

In review, it is generally agreed by many writers that the following factors are important in the perforation of the bile duct system: (*a*) increased intraductal pressure due to obstruction or spasm of the sphincter of Oddi, or to both; (*b*) necrosis of a portion of the bile duct secondary to calculus; (*c*) infection with necrosis of the wall; (*d*) thrombosis of the mural vessels with necrosis of part of the ductal wall; (*e*) diverticula and cysts, and (*f*) activation of reflux pancreatic secretions.

In our case in which the only obvious pathology was a gall bladder showing evidence of acute and chronic cholecystitis, we could conjecture that frequent attacks of infection had invoked some cholangitis with resultant attenuation of the wall, a combination of factors (*a*), (*b*), and (*d*) of the preceding paragraph. That the accompanying spasm of the sphincter of Oddi played a part, we believe, is conceivable. The role played by reflux of activated bile or by the presence of infection unfortunately cannot be evaluated.

Rupture of any portion of the ductal system leads to peritonitis. When the perforation is confined to the common duct, the majority of cases may be microscopic rather than gross rupture. Gariepy and his co-workers [14] feel that since choledochal calculi are relatively frequent, their role cannot be underestimated.

SUMMARY

A case of spontaneous perforation of the common duct with biliary peritonitis in a 65-year-old man, with recovery, is reported. A cholecystectomy, with drainage of the common duct and abdomen, was performed. Early surgical intervention is imperative with due regard to preoperative preparation and correlative laboratory work-up. This condition is rare and is presented with a general review of the literature.

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RETROPERITONEAL DUODENAL RUPTURE DUE TO BLUNT TRAUMA

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THE SIGNS and symptoms produced by a free perforation of a hollow viscus into the peritoneal cavity are ordinarily so typical that the diagnosis is easily suspected. However, retroperitoneal rupture of a similar viscus initiates relatively obscure symptoms and bizarre physical findings. It is not surprising, therefore, that retroperitoneal perforations of the duodenum are perplexing diagnostic problems. They are usually not diagnosed before surgical exploration and may even be overlooked at operation. In addition, these lesions are formidable problems in surgical management. For these reasons, two such cases seen at this clinic seem worthy of addition to the surgical literature.

One hundred thirty-four cases of nonpenetrating traumatic rupture of the duodenum from the world literature prior to 1910 were collected and reviewed by Guibé.[1] Eighty-four additional cases between 1910 and 1940 were collected by Hinton.[2] The mortality in these cases was reported as 90 and 45%, respectively. Siler [3] has recently reported four cases, all of whom survived. An indication regarding the incidence of subcutaneous duodenal rupture as compared with the frequency of other nonpenetrating traumatic intestinal perforations is furnished by Cooke [4] and by Counseller and McCormack,[5] who give similar figures, of about 9%. According to Lauritzen,[6] free traumatic duodenal perforation occurs about twice as frequently as retroperitoneal rupture due to blunt trauma.

These nonpenetrating duodenal injuries occur with varying frequency in different parts of the duodenum. The commonest location is at the junction of the second and third portions of the duodenum. The duodenojejunal angle is the second most frequent site of rupture. This follows the concept that traumatic intestinal perforations occur at or near points of anatomic fixation. Susceptibility of the duodenum to injury by blunt trauma is attributable to its relative fixation at the pyloric and jejunal ends and to the overlying peritoneum, both of which limit displacement from the vertebral bodies. Were it not for its deep location and the protection afforded by the costal margins, the liver, and the transverse colon, injuries to the duodenum would undoubtedly be commoner.

While abdominal signs and symptoms occur promptly and are usually generalized after free perforations of the duodenum, they may be delayed or limited to the right or left abdomen following retroperitoneal perforations, depending on which side of the retroperitoneal space the leakage occurs.

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Up to 30% of suspected retroperitoneal perforations reported in the literature were not located at the time of surgical exploration. This should emphasize the necessity of adequate and meticulous exposure of the retroperitoneal portion of the duodenum in suspected cases following trauma.

Butler and Carlson [7] report on a patient who walked home after an automobile accident only to be hospitalized nine hours later with upper abdominal pain, bilateral testicular pain, and palpable perirectal emphysema. The roentgenographic finding of a perirenal gas shadow which also outlined the right psoas muscle and cecum was reported by Sperling and Rigler.[8] They also noted the presence of a residual deformity of the second part of the duodenum during a barium study five months after injury.

Thus, a relatively silent interval following abdominal trauma and the paucity of early diagnostic signs may belie the presence of retroperitoneal duodenal rupture and obscure the urgent indication for surgical intervention. Delay in onset of fever, elevation of pulse rate, and leucocytosis are further deterrents to accurate diagnosis.

Patients with traumatic retroperitoneal duodenal perforations may be seen either shortly after injury or days or months later after complications have arisen. The surgical problem presented by such cases depends largely upon the length of this interval and the complications present.

CASE 1.—W. H. M., 487217, a 23-year-old white man, was admitted to the University Hospital on Aug. 3, 1948, seven hours after he had sustained abdominal trauma. He related that when the truck which he was driving struck a tree stump, he was thrown forward, striking his upper abdomen against the steering wheel. After a transient period of unconsciousness, he experienced moderately severe, right midabdominal pain and repeated vomiting but was able to walk about 50 yd. (45.5 meters) to the road. A passing motorist took him to a nearby hospital, where he was admitted and observed for three hours. During this interval, his physician reported the onset of minimal right abdominal rigidity and tenderness without evidence of shock. He was then administered ½ grain (30 mg.) of morphine and transferred 70 mi. (112 km.) by ambulance to this hospital. On arrival, the abdominal pain had not radiated or changed in severity or location.

On physical examination the temperature was 98.8 F., the pulse rate 104, respiration rate 16, and blood pressure 130/80. The significant physical findings were confined to the abdomen. It was scaphoid and moved freely with respirations. Liver dullness was present. Moderate tenderness and rebound tenderness were found in the right paraumbilical area and to a slighter extent in the right lower quadrant. No epigastric, subcostal, or rectal tenderness was elicited. Bowel sounds were not audible.

The admission laboratory studies included a hematocrit reading of 50%, leucocyte count of 13,700, with 98% total neutrophiles; a serum amylase determination reported as 198 Somogyi units, and normal urine.

Upright, recumbent, and left lateral decubitus roentgenograms of the abdomen demonstrated no pneumoperitoneum, but the right psoas shadow was absent.

Since no specific indication for emergency surgical intervention was present, Levin-tube suction was instituted, and the patient was observed overnight. During this period his temperature rose to 102 F. despite hydration, and the points of maximum and rebound tenderness descended farther into the right lower quadrant.

Eighteen hours following injury, with preoperative diagnosis of probable perforated viscus, celiotomy was performed through a right midparamedian incision. When the peritoneal cavity was entered, a small amount of odorless brown-tinged fluid was encountered. Exploration of the viscera failed to demonstrate any obvious free perforation. However, the serosa of the ascending colon was noted to be elevated by air bubbles and bile-tinged fluid. This finding prompted mobilization of the ascending colon and hepatic flexure medially to inspect the posterior aspect of the colon and the duodenum. As the duodenum was mobilized medially, a

posterior retroperitoneal transverse laceration was found to involve about half of its circumference in the distal section of the third part adjacent to the vertebral bodies. No other injury was found. The laceration was closed transversely with two layers of interrupted nonabsorbable surgical (silk) sutures. In addition, a complementary Stamm-type jejunostomy, utilizing a No. 16 F. catheter, was performed 16 in. (41 cm.) distal to the ligament of Treitz. The catheter was passed proximally to a point just above the duodenal closure and was used for suction and decompression during the first four postoperative days. On the 5th day, the catheter was retracted into the jejunum, where it was used for jejunostomy feedings until its removal, on the 10th day. Recovery was uneventful. An upper gastrointestinal series 14 months later showed no appreciable duodenal deformity. The patient had been asymptomatic in the interim.

CASE 2.—G. F. D., 522459A, a 31-year-old white man, was admitted to University Hospital on April 3, 1952, six months following injury in an automobile accident. At the time of injury, he had been thrown forward, striking his upper abdomen against the steering wheel. He stated that he remained unconscious in the automobile for about five hours but was able to walk unaided to a nearby farm house. He was taken to a community hospital where he was examined, and a right Colles' fracture, a lacerated scalp, and multiple contusions of the head, face, and lower extremities were found. The fracture was reduced, his scalp laceration was sutured, and he was under observation for two days, until he signed his own release and returned home.

Within four days of the accident he developed nausea, progressive weakness, left lower quadrant pain, and hematemesis. These symptoms were progressive and necessitated his admission to another hospital five days following injury.

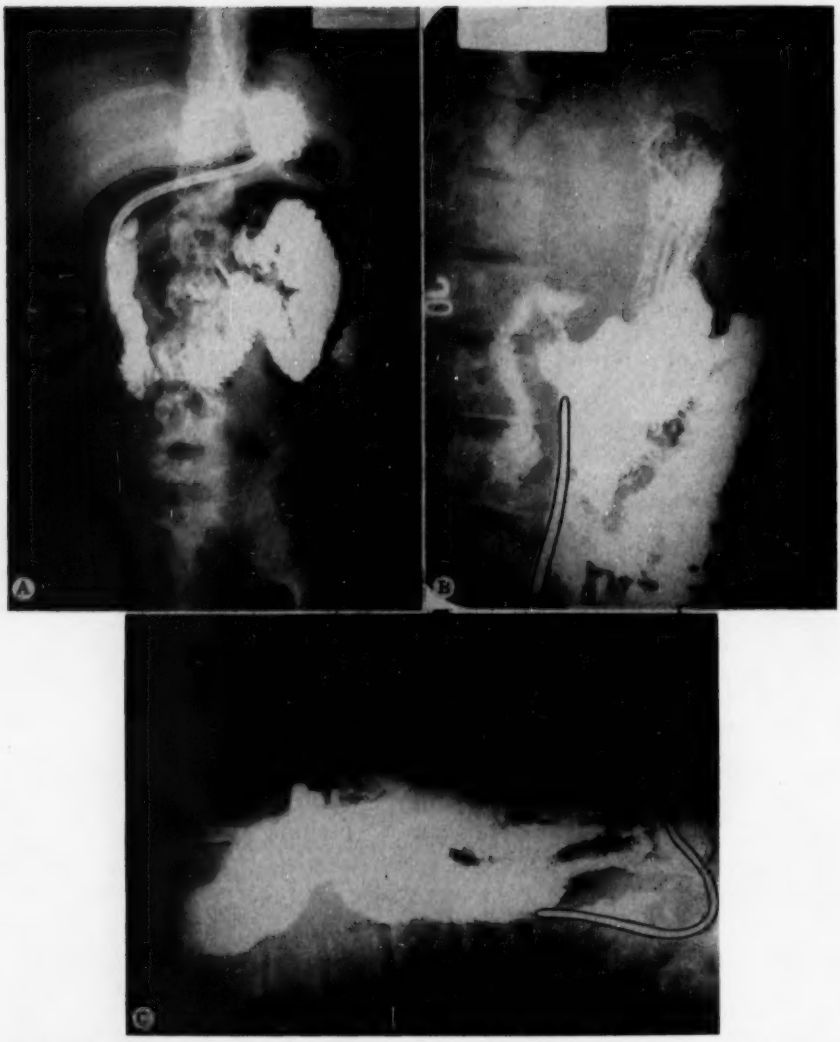
During his second hospitalization, the left lower quadrant pain, described as intermittently sharp-stabbing and dull-aching, radiated to the left thigh and was aggravated by walking and somewhat relieved by left hip flexion. He was given no further definitive therapy and was discharged after 10 days to return to work. However, the left lower quadrant pain, nausea, and vomiting recurred intermittently. By early December, 1951, these symptoms became so severe and frequent that he was incapacitated. This necessitated return to his parents' home in Columbus, Ohio, where the same symptoms continued and his left thigh became relatively fixed in flexion. Attempts to walk caused excruciating left lower quadrant pain. He experienced intermittent bouts of diarrhea and became progressively more debilitated with continuing weight loss. He was seen on several occasions by his family physician, who recommended hospitalization, which he refused. A more acute three-day illness initiated by nausea, diarrhea, and progressing to vomiting, distention, and obstipation finally prompted him to request hospitalization. He had lost 50 lb. (23 kg.) in the interim of six months since injury.

On admission his blood pressure was 120/70, pulse rate 120, and respiration rate 24. He was an acutely ill, markedly dehydrated, cachectic, young man, whose left thigh was acutely flexed. Pertinent positive physical findings were confined to the abdomen and the left hip. The abdomen was moderately distended, tympanitic, and diffusely tender. Slight rigidity was noted in the left lower quadrant, where a large, exquisitely tender, boggy mass, about 10 cm. in diameter, extending to the inguinal ligament was palpable. Bowel sounds were hyperactive, but no peristaltic rushes were audible. Attempts to passively extend the left hip initiated severe left lower quadrant pain. The left lower quadrant mass was also palpable and tender on digital examination of the rectum. The admission diagnosis was pancreatic pseudocyst or a retroperitoneal abscess.

Significant admission laboratory determinations included an erythrocyte count of 3,720,000; hemoglobin content, 7.6 gm. per 100 cc.; leucocyte count, 19,000; blood urea nitrogen, 45 mg. per 100 cc., and serum amylase, 277 Somogyi units. In addition, the amylase value of aspirated peritoneal fluid was reported as 430 units.[9] It was apparent, therefore, that the patient had concurrent acute pancreatitis. For this reason, and because he was improving rapidly with conservative management, drainage of the left lower quadrant mass was postponed.

After hydration, electrolyte replenishment, and blood transfusions, his temperature increased to 102 F. and his pulse rate decreased to 88.

A needle aspiration of the left lower quadrant mass three days following admission was productive of bile-stained pus which contained over 400 Somogyi units of amylase. These findings suggested the presence of a retroperitoneal abscess originating from a retroperitoneal traumatic perforation of the duodenum. By this time, the abdominal distention had responded



Roentgenograms of upper gastrointestinal tract, demonstrating deformity at duodenojejunal angle (*A*). A catheter introduced into upper extent of cavity at surgery is shown lying just posterior to duodenal deformity (*B* and *C*).

to gastric intubation, and the patient began having normal stools. An upper gastrointestinal series was obtained which demonstrated a deformity and partial obstruction at the duodenojejunal angle (Fig. *A*). His condition rapidly improved with supportive and antibiotic therapy. On the 11th hospital day an extraperitoneal drainage of the retroperitoneal abscess was performed through a left lower quadrant muscle-splitting incision. Digital examination of the abscess cavity indicated that it extended superiorly and medially as well as into the left pelvis. A catheter introduced into the upper extent of the cavity at surgery was subsequently shown on roentgenograms to lie just posterior to the previously demonstrated duodenal deformity (Figs. *B* and *C*).

The patient was ambulatory and taking a high-caloric, high-protein diet on the first postoperative day. During his last week in the hospital the limitation of motion of the left hip disappeared and drainage from the wound ceased. Injection of radiopaque solution through the catheter before its removal reaffirmed the intimate relation between the upper extent of the abscess cavity and the terminal duodenum. He was discharged on the 25th hospital day, having gained 17 lb. (8 kg.) since admission. He has since returned to work and has gained an additional 12 lb. (5 kg.).

TREATMENT

The operative management of patients seen within 24 hours following retroperitoneal perforations of the duodenum should consist of adequate duodenal exposure and closure of the perforation. In addition, a Stamm-type jejunostomy, utilizing a Levin tube inserted about 18 in. distal to the ligament of Treitz and threaded proximally into the duodenum or a duodenostomy, should be employed. The jejunostomy allows duodenal decompression until the patient begins to have stools. Later, the catheter may be withdrawn into the jejunum to allow institution of jejunostomy feeding.[10] Thus, postoperative leakage at the site of closure may be obviated. The jejunostomy should not be abandoned until it becomes apparent that duodenal leakage or fistula is not likely to occur.

If the patient is seen at a longer interval after injury with a retroperitoneal abscess, the lesion should be drained extraperitoneally at the most accessible point. In the event that a duodenal fistula with a significant volume of drainage is present, or results after the evacuation of a retroperitoneal abscess, a jejunostomy is indicated in order to feed exclusively by this route. After jejunostomy and proper jejunostomy feeding, the duodenal fistula is likely to close spontaneously.

SUMMARY

Retroperitoneal rupture of the duodenum produces equivocal signs and symptoms less characteristic than free intestinal perforations. Although this injury occurs rather infrequently, it produces a high mortality and is a difficult surgical problem in both diagnosis and management. Retroperitoneal rupture of the duodenum seen within 24 hours of injury should be treated by primary closure and complementary jejunostomy. Jejunostomy decompresses the duodenum and may subsequently be utilized for feeding.

If the patient is seen later with an abscess following retroperitoneal rupture of the duodenum, extraperitoneal drainage should be performed. A jejunostomy and exclusion of oral intake should also be utilized if a significant fistula is present or follows drainage of an abscess. Repair or resection is indicated only when jejunostomy is not followed by spontaneous closure and the patient's status has been sufficiently improved.

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CLINICAL EXPERIENCE IN INTRAVENOUS ADMINISTRATION OF COCONUT WATER

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THE CLINICAL, hematologic, and antigenic properties of coconut water have been described earlier.* The purpose of this paper is to review our clinical experience with the intravenous administration of coconut water in 157 patients and to evaluate its clinical role as an intravenous solution for use in parts of the world where pyrogen-free solutions cannot be obtained.

MATERIALS AND METHODS

Fresh coconut water was administered intravenously to patients according to the methods previously discribed.*

The patients used in this study were from the surgical wards of the Siriraj and Chulalongkorn Medical School Hospitals in Bangkok, Thailand (4 patients); from the surgical wards of the Barnes Hospital in St. Louis, (17 patients), and from the wards of the Tela Railroad Hospital in Tela, Honduras (136 patients). These patients were well hydrated, had a good urinary output, and were not acutely ill at the time of infusion.

The coconuts used in Thailand and Honduras were obtained locally. Those used in St. Louis were obtained from Miami, Fla.† Only clear fluid of low-fat content from the immature nut was employed in this study.

Three methods were utilized for treating coconut water prior to its infusion intravenously (Table 1). A group of eight patients received coconut water autoclaved at 15 lb. (6.8 kg.) pressure for 20 minutes prior to its administration. Another group of 71 patients received coconut water to which 100,000 units of penicillin G had been added. The final group of 75 patients received untreated coconut water as it was withdrawn from the fruit. The only precaution taken in the latter group was the removal of large particulate matter by filtration through a few layers of sterile surgical gauze.

The temperature, pulse, respiratory rate, and clinical appearance of all patients were followed during the infusion and for at least 24 hours thereafter.

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Part of the expenses involved in this study were defrayed by the United Fruit Company.

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* References 6 and 7.

† Through the courtesy of Mr. A. D. Barnes, Director, Dade County Park Department.

The rate of administering coconut water varied widely. At first, a slow rate of infusion was employed (4 drops per minute), but as confidence in the technique was gained, the rate was increased. The infusion was regularly started at a rate of 15 drops per minute in order to minimize any sensitivity reaction that might develop. When no reaction appeared within five minutes, the rate of injection was increased to about 5.2 ml. per minute. Using this procedure, a 500 ml. infusion was given in slightly over one and one-half hours (Table 3).

TABLE 1.—*Reactions in Patients Receiving Intravenous Coconut Water Analyzed as to Method of Preparing Infusion*

Method of Preparing Fluid	Patients Infused, No.	Reactions, No.	Reactions, %	Remarks
Untreated	78	5	6.4	
Autoclaved	8	1	12.5	Single reaction occurred in sample inadvertently autoclaved twice
Penicillin	71	5	7.0	
Total cases	157	11	7.0	

TABLE 2.—*Case Breakdown of Reactions in 157 Patients Receiving Coconut Water Intravenous Infusions*

Case No.	Reactions	Remarks
1	Fever and urticaria	
2	Fever 39 C. (102.2 F.), chill	Inadvertently autoclaved twice; solution appeared cloudy
3	Chill, severe headache	Appeared only after 1,550 ml. had been administered during 7½-hr. period
4	Slight transient urticaria	No fever or other reactions
5	Chill and fever	
6	Chill and fever	
7	Fever 101 F.	Patient had extensive burns
8	Chill and fever	
9	Chill and fever 38.3 C. (100.94 F.)	
10	Chills, fever, nausea	Used old rubber tubing, probably contained pyrogens
11	Chills, fever 38.3 C. (100.94 F.), tingling hands	
Total	Fever, 10 Urticaria, 2 Headache, 2 Tingling hands, 1	

TABLE 3.—*Rate of Administration of Intravenous Coconut Water to 157 Adults*

	Rate per Minute	Time Required for 500 ml. Infusion
Average	5 to 2 cc.	1 hr., 30 min.
18 patients	Over 8 cc.	Less than 1 hr.
Maximum	16.6 cc.	30 min.

Intradermal injection of 0.1 ml. of coconut water were given into the forearms of 51 patients who were examined for evidence of sensitivity 30 minutes and 24 hours after the injection. Four of these patients were from Bangkok, 20 from St. Louis, and the remaining 27 from Honduras.

Multiple infusions were given to 18 patients. The second infusion was given to six persons within 48 hours after the original administration of coconut water. Ten patients received their

second dose from five to six weeks after the first. This is the time-period interval for obtaining the maximum number of antigenic reactions, should they occur. Prior to reinfusion every patient had a skin sensitivity test performed upon him; it consisted of an intradermal injection of 0.1 ml. of coconut water. Any patient manifesting sensitivity was not given more coconut water. Two patients skin-tested six weeks after their original infusion developed a local reaction and were given no further infusion.

Samples of urine were obtained from 32 patients immediately before and again immediately after the termination of the coconut water infusions. The pH of the urine samples was determined in each case by means of litmus or benzidine paper. Twenty-six patients had an aciduria prior to the infusion and therefore could be used for observing the urinary alkalinizing effect of coconut water.

The amount of coconut water employed in each infusion varied, but in all cases was in excess of 250 ml. Routinely we infused 500 ml. The maximum amount of intravenous coconut water given to a patient in one day was 2,365 ml. Eight patients have received over 1,500 ml. within a 12-hour period.

Twenty-four-hour balance studies have been carried out on five patients receiving intravenous coconut water infusions in an effort to determine whether its diuretic properties were sufficient to produce a dehydrating effect. No attempt was made to control carefully the rate of fluid administration in this section of the study.

RESULTS

Rate of Reaction.—Of the 157 patients receiving intravenous infusions of coconut water, 11 had reactions that might be ascribed to the infusion—a reaction rate of 7%. Three patients had a mild transient pyrexia (maximum of 38.3 C. [100.9 F.]) that might have been caused by factors other than the infusion. One had extensive burns; the second received an infusion through dirty rubber tubing and later had no reaction when the donor set was changed, and the third received coconut water that had inadvertently been autoclaved twice and had a turbid appearance. If these three cases were subtracted from the total number of reactors, a corrected reaction rate of 5.0% would be obtained. Table 2 gives a more detailed analysis of the 11 cases in which a reaction was recorded. Ten of these patients had fever; two had urticaria; two complained of headache, and one woman complained of tingling in her hands. None of the reactions were serious. Only one patient (Case 3, Table 2) was pyretic for more than 12 hours.

There was no significant difference in the reaction rate between the group receiving untreated coconut water and those receiving infusions to which penicillin had been added. Too few patients received autoclaved coconut water infusions to warrant any conclusions regarding the effect of autoclaving upon reaction rate. The cloudy appearance of the fluid following autoclaving and the one case in which a reaction occurred when the fluid had been autoclaved twice suggest that heat denatures the protein of the fluid. For these reasons we discontinued heat sterilization of the solutions prior to infusion.

There was no correlation between the rate of infusion of coconut water and reactions. Eighteen patients received infusions of 500 ml. of coconut water at an average rate of 8 ml. per minute without reaction. One patient showed no untoward effects while receiving a 400 ml. infusion at a rate of 16 ml. per minute.

Pain Along the Infused Vein.—An aching pain was experienced along the course of the vein when the fluid was infused rapidly. This aching pain disappeared as soon as the infusion was slowed. We thought initially that the pain was due to the acidity of the solution,[6] but later realized that it was more likely due to the high potassium concentration of the infusion. The critical rate of infusion that produced distress

along the infused vein varied remarkably from patient to patient. One of the factors was the size of the vein into which the infusion was running. Fast infusions into small veins on the dorsum of the hand were regularly painful, while fast infusions into the antecubital vein were less frequently so.

Cardiac irregularities, palpitation, and hypotension did not attend any infusions, although we carefully looked for them.

Twelve patients were skin-tested for sensitivity five to six weeks following their original infusion. Ten showed no reaction and subsequently tolerated another infusion of coconut water without incident. Two of this group showed a positive skin

TABLE 4.—*Results of Reinfusions of Coconut Water Five to Six Weeks After Original Infusion*

No. Cases	Skin Tests	Remarks
2	Positive	No reinfusion given
10	Negative	Reinfusion given without reaction

TABLE 5.—*Reaction of Urine Immediately Following Coconut Water Administration**

Reaction	No. Cases
Alkaline	23
Neutral	2
Acid	1
Total cases tested	26

* All cases had aciduria prior to infusion.

TABLE 6.—*Diuretic Effect of Intravenous Coconut Water Infusion*

Case No.	Coconut Water Intake Ml.	Urinary Output Ml.	Difference *
1	2,365	2,750	— 385
2	1,975	1,995	— 20
3	2,000	3,240	—1,240
4	2,000	2,610	— 610
5	2,000	3,120	—1,120

* Negative water balance would include an additional daily insensible water loss of approximately 1,000 ml.

test and were given no more. Six patients received one or more reinfusions within 48 hours after the original administration of coconut water; not one had any reaction (Table 4).

Twenty-three of the 26 patients who had an aciduria prior to the infusion of coconut water developed a transient urinary alkaline tide following it (Table 5). Only one patient receiving infusions of this acid solution excreted an acid urine during the immediate postinfusion period.

Eight patients received infusions of 1,000 ml. or more. Two of these patients (Cases 1 and 3, Table 2) developed fever, urticaria, and headache after more than 1,500 ml. of the fluid had been administered.

The results of the water balance studies carried out on patients receiving intravenous coconut water as their only source of fluid for 24 hours are presented in Table 6. A diuresis occurred during every infusion; in fact, the total urinary output during

the infusion of the fluid exceeded the amount infused. The actual negative water balance is more marked than the difference between the intake and output when insensible water loss is taken into account.

COMMENT

It is apparent from our experience as well as others ‡ that coconut water can be administered intravenously without frequent or serious evidence of toxicity. Fresh coconut water is sterile,[7] so treatment of the fluid with penicillin or by heat sterilization prior to infusion is unnecessary.

The aching pain occasionally noted in this series along the course of the infused vein has been described with infusions of other fluids of high potassium content.§ Although the potassium content of coconut water is high (40 mEq. per liter), we have found no evidence of potassium poisoning among the patients included in this study. This may in part be due to the high magnesium content [7] of coconut water which acts as a physiologic antagonist to potassium.‖ None of the patients included in this study were oliguric at the time of infusion. It should be emphasized that intravenous coconut water should be employed only with great care in patients with diminished renal function in order to avoid potassium intoxication.

The urinary alkaline tide noted following administration of strongly acid coconut water is similar to that found after the intravenous administration of potassium chloride.¶ This seeming paradox is due to the rapid renal excretion of potassium,# compared to the less prompt elimination of chloride. Since chloride cannot immediately be excreted with potassium, bicarbonate ions are excreted instead, producing a urine of high potassium bicarbonate content which is alkaline in character despite the acidic nature of the intravenous infusion.

The diuretic effects of intravenous coconut water have previously been noted,* but neither the cause nor the extent of diuresis has been quantitatively measured.

It is of prime importance in the clinical evaluation of coconut water to determine whether the intravenous administration of this material results in an absolute dehydration of the body to a water content lower than preinfusion levels. Substances of this nature are called ecuretic diuretics. Although further water balance studies must be made in patients receiving intravenous coconut water, our evidence would indicate that intravenous coconut water is an ecuretic diuretic and should not be utilized in an attempt to hydrate a patient unable to take oral fluids. We had hoped that coconut water might be employed as a parenteral source of water, but if the present data on water balance are confirmed, its diuretic properties preclude its use as a source of water. In other words, the administration of coconut water intravenously is attended by dehydration, not hydration.

One of the factors in coconut water producing diuresis is its potassium content. Wolf [32] has found that potassium salts are ecuretic only when administered in concentrations above 70 mEq. per liter. This figure, which he describes as the limiting isorrheic concentration [31] for potassium, is far greater than the maximum concentra-

‡ References 4, 21, 17, and 26.
§ References 8, 10, 18, and 22.
‖ References 16 and 18.
¶ References 13, 14, 30, and 31.
# References 2 and 19.
* References 4, 17, 21, and 26.

tion found in coconut water. Normal man can produce urine with potassium concentrations of 300-400 mEq. per liter, a figure some 10 times that found in coconut water. It seems evident, therefore, that there is some substance in coconut water other than potassium that accounts for its marked diuretic effect.

There is a relatively high magnesium concentration in coconut water. Signs of magnesium poisoning, such as weakness, anesthesia, progressive bradycardia, and respiratory depression,† did not appear in the patients included in this study. This is probably due to the fact that all of these patients had an adequate urinary output and eliminated the magnesium in this manner.‡

A clinical evaluation of intravenous coconut water administration requires some mention of its high fructose content.[7] Although there is some question of detail concerning its benefits,[1] fructose appears to be more quickly and efficiently utilized as a source of calories than does intravenously administered glucose.§ With a carbohydrate content of 4 to 7 gm. per 100 cc., fat-free coconut water furnishes an efficient source of up to 240 calories per liter of fluid.

SUMMARY AND CONCLUSIONS

Coconut water has been administered intravenously to a series of 157 patients.

There were 11 reactions in this series of infusions—a reaction rate of 7%. The reactions were transitory and of no serious clinical significance.

Intravenous readministration of coconut water to a series of 12 patients five to six weeks after the initial infusion has produced no evidence of sensitization.

The clinical implications of the high concentrations of potassium and magnesium in coconut water have been discussed. No evidence of toxicity ascribable to these cations has been noted.

When coconut water is infused at a rapid rate, a majority of patients will complain of some discomfort along the course of the infused vein due to the high potassium content of the fluid. When the infusion is slowed, this discomfort disappears.

The data available to us at this time indicate that the diuretic action of coconut water given intravenously is so marked that actual dehydration follows its infusion.

On the basis of present evidence, the diuretic effect of this material makes it of no clinical value in improving depleted states of hydration of a patient. It can be used as a source of potassium, a source of calories, or as a vehicle for the injection of sodium salts added to it for the emergency treatment of hypovolemia when other sterile pyrogen-free fluids are unavailable.

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COMPARATIVE REPORT OF PARTIAL GASTRECTOMY AND VAGOTOMY IN TREATMENT OF PEPTIC ULCER

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WHEN ONE applies gastric resection or the Dragstedt operation to the surgical treatment of patients with ulcer disease, it must inevitably be concluded that vagotomy has objective advantages over partial gastrectomy. Vagotomy is in harmony with the neurovegetative pathogenesis of ulcer, gives a lower mortality, leaves the anatomic unity of the stomach untouched, and technically is easier to perform. A further important advantage of vagotomy is the decreased postoperative morbidity.

Gastric resection, on the other hand, results in a high mortality rate, especially if performed in a surgical department staffed by a large number of surgeons. Thus, Quenu of Paris has reported a mortality of 6.6% in the performance of 500 gastric resections, and Huber of Vienna has encountered a mortality of 7.2% for 2,993 cases. This means that at Quenu's clinic every 15th patient died and at Huber's every 14th patient succumbed after partial gastric resection. If to this high mortality one adds the very well known complications occurring in the early and late postoperative periods, such as the recurrence of ulcer or pain, or the early and late postprandial syndromes, then one comes to the conclusion that the problem of surgical treatment of ulcer is not satisfactorily solved by gastric resection.

In contrast, Dragstedt and Woodward have reported a mortality rate of 0.9% in a series of 509 vagotomies. That vagotomy with gastroenterostomy is attended by a small proportion of postoperative complications is supported by a study of the American Gastroenterological Association. In 1,046 patients undergoing vagotomy and gastroenterostomy, satisfactory results were obtained in 93.2% of the cases, unsatisfactory results in 5.3%, and recurrence of ulcer took place in 1.5% of the cases.

From a total of 128 partial gastrectomies carried out from the beginning of 1947 until the end of 1952, a follow-up study of 110 patients revealed the results shown in Table 1.

These data indicate that 41.7% of the patients have severe symptoms after gastric resection and that 7.2% have died as a consequence of the operation. Thus, the unpleasant fact is that almost one-half of the patients with resection must be considered either as lifelong invalids or as lost. It would seem that in critiques favorable to partial gastrectomy insufficient attention is paid to abdominal pain, anemia, recur-

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rence of ulcer, and failure of the patient to maintain an adequate nutritional status. Too much emphasis has been placed on a progressive lowering of mortality rates in arriving at the conclusion that resection of two-thirds of the stomach is a benign procedure.

From the beginning of 1947 until the end of 1952, vagotomy was performed on 173 patients. In the beginning of that period, vagotomy alone was done. This group included 53 patients, and only 37 of these have been available for follow-up studies.

TABLE 1.—*Results in 110 Patients Treated by Partial Gastrectomy*

Results	Patients	
	No.	%
Excellent	56	50.9
Postprandial syndrome	25	22.7
Recurrence of pain	18	16.3
Recurrence of ulcer	3	2.7
Early death in connection with operation	6	5.4
Late death in connection with operation	2	1.8

TABLE 2.—*Results in 116 Patients Treated by Vagotomy with Gastroenterostomy*

Results	Patients	
	No.	%
Excellent	108	93.1
Malfunctioning of anastomosis	3	2.5
Severe gastritis	4	3.4
Death at later date without connection either to ulcer or to operation	1	0.8

TABLE 3.—*Results of Vagotomy and Gastroenterostomy Compared with Results of Gastrectomy*

Operation	Patients					
	Total	Follow-Up	Early Death	%	Late Complications	%
Vagotomy & Gastroenterostomy	120	116	..	..	7	5.9
Gastric Resection	128	110	6	5.4	46	41.7

Among them, 13 patients had recurrence of symptoms. Thus, with unsatisfactory results in 35% of these patients, simple vagotomy was discontinued except in treatment for gastrojejunal ulcers. Thereafter, vagotomy combined with gastroenterostomy was carried out in 120 patients.

From a total of 120 patients treated by vagotomy with gastroenterostomy, follow-up studies on 116 patients revealed the results shown in Table 2.

As these data indicate, there were no operative deaths. In a later period, remote from operation, one patient developed a pulmonary abscess and died. It may be seen that the percentage of unsatisfactory results is 5.9%. Of the three patients with malfunctioning of their anastomoses, two were reoperated on with the construction of new gastroenterostomies and subsequently felt well. The third patient underwent resection at another hospital, the outcome of which is not known.

Gastritis, after gastroenterostomy, is probably due to the presence of bile and pancreatic juice in the stomach. Of the four patients with gastritis, one developed massive bleeding which required partial gastrectomy, another is an alcoholic, and the last two patients have continued to have signs of gastritis as judged by clinical and radiologic tests.

Therefore, in view of the small percentage of unsatisfactory postoperative results, the absence of operative deaths, and the maintenance of anatomic unity of the stomach, the authors prefer vagotomy with gastroenterostomy as the surgical treatment of choice for chronic, callous, nonpenetrating duodenal ulcer.

This opinion is very convincing when compared with the results obtained by gastric resection (Table 3).

CONCLUSIONS

Late, comparative results of six surgeons from the same department of surgery are given for 116 patients receiving vagotomy and gastroenterostomy and for 110 patients receiving gastric resections in treatment for peptic ulcer.

Particularly, it is pointed out that there were no deaths in the 116 patients treated by vagotomy, while there were six deaths in the immediate postoperative period following gastric resection and two additional deaths within several weeks following the operation.

Of the 116 patients treated by vagotomy and gastroenterostomy, 93% had excellent results. Of the 110 patients receiving gastric resection, 51% had excellent results.

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PRESERVING ARTERIAL SEGMENTS FOR THE BLOOD VESSEL BANK

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DETROIT

SINCE vascular surgery has come into prominence, there has been an increasing demand by surgeons for blood vessels to use as grafts. The first blood vessel grafts were obtained from the patients themselves or were vessels freshly obtained at autopsy performed immediately after death. This method is unwieldy and impractical for even the largest institutions, since the opportunity for obtaining a suitable vessel rarely coincides with the opportunity for using it.

To obviate this incongruity, methods of preserving blood vessels were investigated and blood vessel banks were established. Many of the methods of preservation and storage have the drawbacks that the blood vessels must be obtained within a short time after death and the preserved vessels must be used within a limited period, making them impractical for any but the larger hospitals.

In the past several years we have used freezing, storage in physiological solutions, and lyophilization as methods of preservation. Of these we chose lyophilization as the most practical and satisfactory method. It lessens the storage problem by a large degree and prolongs the storage period from a few weeks to several years.

PROCEDURE

Human and animal aortas were used in these experiments. The aortas were obtained at autopsy between 2 and 36 hours after death. If the autopsies were not performed immediately, the bodies were refrigerated. The aortas were removed with clean, nonsterile instruments before the rest of the viscera were disturbed. Gloves were worn, but not masks. To aid in later selecting a blood vessel of the proper size, the length and diameter of the blood vessels were recorded upon removal since lyophilization distorts the vessel.

The blood vessels were lyophilized as described by Pate [1] and Jordan.[2] They were placed in a large test tube, which in turn was placed in a vessel containing dry ice and acetone for rapid freezing. The tube containing the blood vessel was connected to a vacuum pump capable of reducing pressure to 10 microns in minutes. The vacuum was maintained for 24 hours.

When the aorta had been dehydrated it was placed in a polyethylene envelope, which was then heat sealed. Any air present was evacuated with a syringe and the needle hole resealed.

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Nitrogen could be injected to insure a complete lack of air. The envelopes containing the aortas can now be stored as they are at room temperature. They are unbreakable, and they require but a small space for storage.

The aortas may be sterilized at this time or they may be sterilized just before use. Sterilization just before use permits the sterilization of a number of aortas at the same time. Using data compiled by Moriarity,[3] we exposed the envelopes containing the aortas to an electron beam in the Van de Graaff electron accelerator and administered a calculated dose of 4,000,000 rep (83 ergs per gram).

RESULTS

After the grafts had been lyophilized and sterilized, they were cultured and found to be sterile. Some of the grafts were reconstituted by placing them in Ringer's solution and placed in animals, with no increase in the incidence of thrombosis or blowouts when compared with fresh grafts.

These grafts have also been examined microscopically to determine the amount of tissue disruption from this method of sterilization.

To test the effectiveness of irradiation for sterilizing blood vessel grafts we obtained aortas and intentionally contaminated them with spore-forming organisms and organisms notorious for their resistance to antibiotics. To insure contamination an aorta was placed in a nutrient solution containing Bacillus subtilis and Aerobacter aerogenes. The contaminated aorta was next taken from the nutrient solution and cut into four sections. Two of the four sections were placed in polyethylene bags, which were then heat sealed. One of these two sections received no further treatment. The second section was placed in the Van de Graaff machine for sterilization without being lyophilized. The two remaining sections were lyophilized before being sterilized in the Van de Graaff machine. One of these two sections was then rehydrated in Ringer's solution. All four sections were recultured. Cultures were negative on the three sections which had been exposed to the Van de Graaff machine. The first section, the control, showed the same organisms, B. subtilis and A. aerogenes.

The same procedure was repeated with a number of other aortas contaminated with one or more of these organisms: Aspergillus, Escherichia coli, Proteus, Pseudomonas aeruginosa, and Candida albicans. The results were the same.

No virus cultures were done, but according to the data of Moriarity the viruses require much less irradiation than spore-forming organisms.

Microscopic sections were made of each of the four segments of aorta and they were stained with hematoxylin and eosin. The changes in the irradiated segments were no different from those present in the nonirradiated segments.

Some of the rehydrated lyophilized sections which had been sterilized with the Van de Graaff machine were placed in dogs. The immediate results compared favorably with our control series in which we used sterile lyophilized, nonirradiated aortas. These segments handled like fresh grafts, showing no tendency to tear under suture tension. The tissue was not friable or rubbery, and it showed no other evidence of degeneration.

COMMENT

Fresh, aseptically obtained aortic segments are ideal for use in vascular surgery except that their availability is limited. Therefore it becomes necessary to turn to preserved grafts. In our experimental surgery we have tried several methods besides the lyophilization and found that each had its drawbacks.

When the vessels are frozen with dry ice and acetone without being dehydrated and are stored in a deep-freeze compartment, ice crystals form within the cells. These crystals increase in size and cause disruption of the tissue unless the grafts are used within a very few days. When such grafts are used after several weeks, the percentage of thromboses and blowouts in the immediate postoperative period is very high.

Grafts stored in buffered isotonic solutions (saline, Locke's, Ringer's, or modified Tyrode's solution) all have the disadvantages that the grafts must be used within approximately six weeks, they are very subject to contamination, and they must be obtained by sterile technique within two to six hours after death. Because of the short length of time they can be kept, it is a prodigious task to build up an adequate artery bank by this method. The storage problem is of considerable importance, since they require a great deal of refrigerated space.

The lyophilized grafts obtained with sterile technique eliminate most of the objections present in the other methods of preservation and storage. Since lyophilization stops bacterial activity but preserves any bacteria present in the vessel, all grafts obtained by nonsterile technique are considered contaminated and are exposed in the Van de Graaff electron accelerator to 4,000,000 rep after lyophilization. We feel that with this method of sterilization the interval between the time of death and the time of obtaining the aortic segments can be increased from 2 to 6 hours to 36 hours if the bodies have been refrigerated, thus increasing the supply of material available for the blood vessel bank and eliminating the necessity for sterile technique at autopsy. In addition, it would permit a bank to be built up by permitting the blood vessels to be stored with a greatly increased time limitation.

As most hospitals do not have an expensive Van de Graaff machine, this method will increase in practicality as more radioisotopes are released. The isotopes, such as cobalt, emit gamma rays that are of the same magnitude as the electron beam produced by the Van de Graaff and can be substituted for it.*

Sterilization with the Van de Graaff electron accelerator or with radioisotopes will by their expense be limited to the larger institutions. However, the ease of storage, transportation, and the cessation of bacterial activity after lyophilization would allow the small hospitals to accumulate a number of lyophilized, unsterile blood vessel segments and send them to the larger centers for sterilization.

CONCLUSIONS

Unsterile aortic segments showing no gross changes and taken up to 36 hours after death can be sterilized by irradiation after they have been lyophilized.

The sterilization by means of the Van de Graaff electron accelerator or radioisotopes can be done at any time prior to use.

Lyophilized blood vessels can be stored with ease at room temperature for an indefinite period of time.

On microscopic sections, the blood vessels sterilized by irradiation showed no more degenerative changes than those present in the control vessels which had not been irradiated.

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*Corrigan, K. E.: Personal communication to the authors (from the X-Ray Physics Department, Harper Hospital).

Short-term observation showed no more thromboses or blowouts than in a comparable group of lyophilized grafts obtained in a sterile manner and not exposed to irradiation.

Drs. E. G. Upjohn, W. F. McLimans, N. A. Drake, W. W. Allen, A. A. Humphrey, and P. Jordan assisted in this work

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PANCREATIC CYSTOGASTROSTOMY, A DEFINITIVE TREATMENT FOR PSEUDOCYSTS OF THE PANCREAS

Review of the Literature and Report of Two Cases

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SINCE 1882, when Gussenbauer [17] first successfully treated a pancreatic pseudocyst by marsupialization, these lesions have received exclusive surgical therapy. Numerous basically similar classifications of cysts of the pancreas have been proposed,* but that classification first described by Mahorner and Mattson [19] is the most widely accepted. From this etiological classification of pancreatic cysts, the two groups which by incidence derived by far the greatest prominence in therapy are those cysts resulting from trauma and the neoplastic cysts. Much the commoner disease entity of these two are those cysts resulting from trauma (chemical, physical, or bacterial). Since the bulk of these so-called traumatic cysts (which are devoid of any epithelial lining, and so are not true cysts) have been found unassociated with any history whatever of abdominal trauma, the term pseudocyst has been widely adapted in its place.†

The optimum disposition of the neoplastic cyst of the pancreas is not argued currently. Complete excision of these lesions is the ideal therapy. This group of cysts includes cystadenoma, cystadenocarcinoma, and the teratomatous cysts. When these proliferative cysts are not completely excised, recurrence in one form or another is the rule. Sawyer and his co-workers [29] described a patient in whom a papillary cystadenoma of the pancreas was marsupialized; thereafter there was profuse drainage through the cutaneous fistula for a 10-month period. Two subsequent operations were necessitated to effect a cure, the last procedure being a total excision. This same author, in a survey of the currently reported cystadenomas of the pancreas, was able to find five instances in which frank malignant change occurred in previously benign neoplastic cysts of the pancreas. Carter and Slattery [6] presented a patient in whom a draining sinus persisted for nine years following marsupialization of a proliferative cyst; total excision of this cyst evoked an immediate cure. Numerous other authors strongly advocate the necessity for complete extirpation of these neoplastic cysts of the pancreas.‡

However, concerning pancreatic cystic disease, widest attention has been focused in recent years on the etiology, diagnosis, and treatment of the pseudocyst. Between 40 and 50% of all pancreatic cysts are pseudocysts. These lesions are not common,

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* References 1, 25, 19, and 31.

† References 18, 17, 9, 11, 25, 26, 1, 8, and 31.

‡ References 2, 6, 24, 31, 34, and 27.

as evidenced by a recent report of Collins [9] in which he summarized from the literature 306 proved pseudocysts of the pancreas between the years 1861 and 1949. Other testimony as to the infrequency with which these cysts occur is presented by Adams and Nishijima [1] wherein they record six pseudocysts of the pancreas at the Lahey Clinic between the years 1926 and 1945. Cattell and Warren § subsequently totaled 46 pancreatic cysts at the Lahey Clinic to 1953, of which 30 were classified as pseudocysts. Collins [9] recorded only 3 pseudocysts of the pancreas in 209,813 consecutive cases at the Hollywood Presbyterian Hospital between 1924 and 1949. Rabinovitch and Pines [27] presented 11 pseudocysts of the pancreas in the entire record history of the Jewish Hospital, Brooklyn, up to the year 1942.

The etiology and pathology of pancreatic pseudocysts have received excellent treatment by several authors.‖ It is now obvious that physical trauma plays a much less frequent causative role in the etiology of pseudocysts than was previously thought in that contemporary beliefs focus upon the acute and chronic pancreatitides as being the most frequent causative factor. The known relationship between biliary pathology or biliopancreatic ductal anomaly and pancreatitis leads one to anticipate a high incidence of concomitant biliary tract disease in patients with pancreatic pseudocysts. Such was the experience of Pinkham [26] who reported an incidence of biliary tract disease in 6 of 10 pancreatic pseudocysts. Adams and Nishijima [1] similarly reported three of their six patients with pancreatic pseudocysts had concomitant biliary tract disease. A significant incidence of coexistent biliary tract disease was given by Rabinovitch and Pines.[27] Brilhart and Priestley [5] recorded 9 cases of cholecystitis with stones and 3 cases of choledocholithiasis in a group of 44 patients with pancreatic pseudocysts. (One of the cases reported below had concomitant biliary tract disease, while the other had antecedent biliary tract pathology.)

To be sure, biliary tract disease with pancreatitis does not account for the majority of all cases of pancreatic pseudocysts as the antecedent cause. Actual mechanical trauma, however, appears to play an even more infrequent role as a precursory cause of pseudocysts of the pancreas. For example, Koucky and co-workers [18] reported six cases of pancreatic pseudocysts in which a history of abdominal trauma was obtained in only one case. Brilhart and Priestley [5] recorded an antecedent history of trauma in only 2 of 44 cases, and Pinkham [25] presented 10 pseudocysts in which a history of trauma was elicited in none. Cattell and Warren ¶ noted trauma in the history of but 3 of 46 pancreatic cysts.

After biliary tract disease and abdominal trauma have been ruled out as possible precursory causes, there remains a group of patients in most series of reported cases in which no obvious causative factors can be demonstrated. Many of the authors suggest various etiological mechanisms for this remaining group such as vascular pancreatic necrosis, vascular embolic pancreatitis, indirect pancreatic trauma, alcoholism, and bacterial embolic pancreatitis.

The positive diagnosis of pseudocyst of the pancreas can be made by the pathologist only. The preoperative diagnosis, as pointed out by numerous authors, is suggested by the presence of a palpable mass, usually immobile and most commonly situated in the upper abdomen. The mass is most frequently palpated in the left

§ Reference 34, p. 157.

‖ References 19, 17, 25, 31, 18, 26, 27, and 5.

¶ Reference 34, p. 158.

hypochondrium or in the midepigastrium frequently extending down to and below the umbilicus. The mass is as a rule nontender and only occasionally feels cystic. Cattell and Warren # state such a mass is palpable in about 90% of cases.

The history commonly reveals upper abdominal pain, occasionally referred to the back. An account of antecedent gall-bladder disease, previous pancreatitis, or possibly, abdominal trauma would be helpful. Weight loss is nearly a constant finding. Various dyspeptic complaints are frequently encountered, often associated with excessive eructations and a sense of abdominal fullness. Nausea and vomiting are also commonly recorded in the history. These patients often obtain partial relief of their pain (which is usually constant but is not too infrequently of an occasional colicky nature) by assuming a modified knee-chest position of some sort.

Laboratory studies are frequently helpful in establishing a diagnosis of pseudocyst of the pancreas. Evidence of exocrine pancreatic insufficiency can be established by determining an increase of fat and nitrogen in the stool.[5] Elevated blood amylase determinations are commonly encountered also.* Urinary diastase determinations are occasionally seen to be elevated.†

Jaundice is rather frequently encountered with this disease. This jaundice may be the result of a large cyst of the body or a smaller cyst of the head occluding the distal portion of the common bile duct, either by sheer physical compression or by contiguous inflammatory change. Similarly, coexistent biliary tract disease may coincidently produce obstructive jaundice, for example, wherein the distal common duct is occluded by a gallstone. Judd and co-workers [7] recorded that 10 of their 47 cases gave a definite history of jaundice and 3 additional ones had a questionable history of jaundice. Rabinovitch and Pines [27] stated 3 of their 17 patients had icterus. Korte ‡ noted jaundice in 9 of 121 cases.

Hematemesis and melena of varying degrees have been recorded also in conjunction with pseudocysts of the pancreas.[33] Left hydrothorax has been reported complicating pancreatic pseudocysts by Gross and others,[13] and Edlin.[11] The latter case [11] illustrated the presence of a pseudocyst of the pancreas in the posterior mediastinum, having apparently extended through the esophageal hiatus in the progress of its formation. Diabetes is not uncommonly associated with pseudocysts of the pancreas.§ Brilhart and Priestley [5] reported that 6 of 44 patients were found to have concomitant diabetes mellitus. Portal vein obstruction has also been described in association with pancreatic cysts.[6] Hematuria is another bizarre finding ascribed to pseudocyst of the pancreas,[17] apparently resulting from renal congestion due to partial compression of the renal vein by the cyst.

The highest incidence of pseudocyst of the pancreas is seen between the ages of 30 and 60 years, with the mean age in some large series being in the fifth decade of life.‖ Such cysts have been seen in all age groups, including very early childhood.¶

The most helpful laboratory procedure in establishing a preoperative diagnosis of pseudocyst of the pancreas is the roentgen examination. With a contrast medium in

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Reference 34, p. 165.
* References 5, 26, 25, 18, 31, and 28.
† References 4 and 28.
‡ Cited by Rabinovitch and Pines.[39]
§ References 11, 17, 28, 31. Cattell and Warren,[34] p. 165.
‖ References 5 and 17.
¶ Cattell and Warren,[34] p. 157.

the upper gastrointestinal tract, visible significant encroachments upon, and displacements of, the stomach, duodenum, duodenojejunal juncture, and transverse colon are most helpful, not only in confirming a retroperitoneal space-occupying mass in the region of the pancreas but also in localizing the approximate area of the pancreas which is involved. Numerous illustrative examples of the great help offered by these examinations are recorded.#

In the actual surgical treatment of these lesions, external drainage of the cysts through the skin of the abdominal wall by marsupialization has enjoyed the widest popularity in the past since the first such successful procedure by Gussenbauer * in 1882. The very wide acceptance of this procedure in therapy for pseudocysts is attested to by numerous reports.† The frequent prolonged period of drainage from the pancreatic fistula after marsupialization has been the main complicating factor subsequent to this operation. Kerr ‡ recorded a case in which the fistula drained for 15 years. Considerable quantities of secretion can escape from these sinuses daily, as evidenced by Cushing,§ recording a daily discharge of 500 to 600 cc. from such a fistula. In addition to fluid and electrolyte problems as well as digestive disturbances entailed as a result of these prolonged pancreatic discharges, there can be extremely troublesome skin irritations due to the fermentative actions of pancreatic enzymes upon the skin of the abdominal wall. Secondary infection with abscess formation within the tract and cyst cavity is recorded all too frequently also. Numerous instances of recurrence of the pseudocyst subsequent to marsupialization are on record.|| Cattell and Warren ¶ described recurrent cyst formation or persistent fistula in 7 of 12 patients who were treated initially by marsupialization. Hemorrhage and acute intestinal obstruction have also been recorded as being complications of marsupialization of pancreatic pseudocysts.

In 1923, Jedlicka # first reported anastomosis of the pancreatic duct to the posterior wall of the stomach after excision of a pancreatic cyst. Eight years later, Jurasz * effected a successful transgastric primary cystogastrostomy for pancreatic cyst. In 1926, Gutierrez † reported a cure of a postmarsupialization fistulous tract by implanting this sinus into the stomach. Harries [15] recorded a similar cure of a persisting pancreatic sinus following external drainage of a pancreatic cyst by implantation of the fistulous tract into the anterior surface of the stomach. This surgeon, struck by the success of the implantation of the fistulous tract into the stomach, attempted as definitive treatment an anterior cystogastrostomy upon his next encounter with a pancreatic pseudocyst, in 1934. He performed a curative anastomosis between the cyst and the anterior surface of the stomach near the greater curvature, performing this anastomosis over a short segment of rubber tubing which he passed into the stomach upon completion of the anastomosis. At the time of writing

References 30, 13, 9, 5, 28, 7. Cattell and Warren,[34] pp. 167-171.
* Cited by Judd and others.[17]
† References 17, 25, 5, 18, 33, 27, and 12.
‡ Cited by Judd and others.[17]
§ Cited by Judd and others.[17]
|| References 6, 15, 13, 17, 31, and 21.
¶ Reference 34, pp. 174-175.
# Cited by Brandenburg and others.[4]
* Cited by Brandenburg and others.[4]
† Cited by Judd and others.[17]

in 1934, Harries postulated that it might be a wiser procedure in the future to perform an anterior gastrotomy and through that opening to create a posterior cystogastrostomy, since he noted the posterior surface of the stomach was usually adherent to the pseudocyst of the pancreas. The astuteness of his observations have since been soundly confirmed. Cattell and Warren ‡ have in five cases excised the tract of a persistent pancreatic fistula flush with the communication to the pancreatic duct in the head of the gland and then anastomosed this duct to the stomach.

Despite the obvious success of these and other scattered cystogastrostomies in subsequent years, other methods of cystovisceral anastomosis were attempted. Chesterman § in 1943, recorded apparently the first successful cystojejunostomy done for pancreatic pseudocyst. Since that time, there have been numerous scattered reports of cystojejunostomy.|| In 1948, Gurwitz and Hurwitz [14] were the first, according to the literature, to apply the Roux-en-Y principle of anastomosis to cystojejunostomy. Others have since reported similar successful Roux-en-Y cystojejunostomy.[22] Satisfactory cystovisceral anastomoses have been accomplished after anastomosis of the pancreatic pseudocyst to duodenum,¶ common bile duct,# and gall bladder.*

In 1951 Brandenburg and co-authors [4] collected from the literature 24 cases of pancreatic pseudocyst treated by cystogastrostomy up to that time. The results in this series were uniformly good, there being only one death in that group; that patient died as a result of hemorrhage from a vessel in the cyst wall. Brandenburg [4] also added to the literature one case of his own successfully treated by cystogastrostomy. Since the appearance of that paper, several other case reports of cystogastrostomy have appeared with consistently good results.†

The morbidity and mortality associated with the so-called "ideal" treatment of pseudocysts (excision) are well authenticated. Carter and Slattery [6] recorded surgical laceration of the common bile duct during excision of pseudocysts in two out of a group of six patients. Bowers [3] described a laceration of the left renal vein during excision of the pancreatic pseudocyst in a group of two excisions reported. Collins [9] recorded a case of excision of a pancreatic pseudocyst in which the posterior wall of the midportion of the stomach along the lesser curvature was so adherent to the cyst that it was necessary to excise this portion of the posterior stomach wall in order to effect excision of the cyst. (How simple it would have been here merely to create a curative anastomosis!) Brilhart and Priestley [5] recorded three deaths among six patients who were subjected to complete or partial excision of a pancreatic pseudocyst. (It should be mentioned here, though, that two of these three deaths occurred in patients in whom pancreaticoduodenectomy had been performed as the treatment

‡ Reference 34, pp. 182-185.

§ Cited by Collins.[9]

|| References 1, 26, and 31.

¶ Reference 5. Kirschner, cited by Scovel and Holliger.[42] Kunc, cited by Scovel and Holliger.[42]

Bowers, R. F.: Personal communication to the author. Reference 6.

* Neuffer, cited by Adams and Nishijima.[1] Walzel, cited by Adams and Nishijima.[1] Reference 21.

† References 13, 8, 26, 30, 7. Bowers, R. F.: Personal communication to the author.

for the pseudocyst.) Cattell and Warren,‡ contrarily, have performed excisions (cystectomy, pancreatoduodenectomy, and distal pancreatectomy) with excellent results and no mortality.

The mortality rate incurred in routine excision of a pseudocyst of the pancreas has been reported many times as being extremely high. This is easy to understand when one realizes the intense inflammatory reaction frequently surrounding the pseudocyst and attaching it to the many neighboring vital structures, such as middle colic vessels, common bile duct, portal vein, transverse colon and stomach, aorta, vena cava, superior mesenteric vessels, and renal vessels. True, for the occasional small pseudocyst confined to the body or tail of the pancreas with minimal adherence to contiguous structures, a total excision would seem to be a sound and logical treatment. Unfortunately, however, such situations are seldom found at the operating table in dealing with pseudocysts. Indeed, one only has to peruse the literature and read of the often frustrating search for a cleavage plane between capsule and cyst wall, of repeated profuse hemorrhages at the operating table, of the lengthy tedious dissections in which one or more vital structures are not at all uncommonly injured, to appreciate the oftentimes extremely difficult and dangerous technical aspects associated with excision of a pseudocyst.

It is indeed fortunate that most proliferative cysts of the pancreas do not have quite the pericystic inflammatory reaction found in pseudocysts, since there is no question as to the only logical sound therapy for such neoplastic cysts, this being complete excision. Marsupialization, internal drainage, or partial excision of these neoplastic cysts allows a residual tumor-lined structure to remain which is capable of malignant degeneration or of persistent drainage which obviously would not cease until the epithelium-lined structure was completely ablated.

The fact that some definitive surgery must be applied to pseudocysts is well attested to by the numerous reports on spontaneous complications of untreated pseudocysts, such as have been mentioned above, these being obstructive jaundice, hemorrhage, extreme malnutrition, progressive pain, diabetes, and a very lethal peritonitis which may occur as a result of either spontaneous or traumatic rupture of these cysts, containing, frequently, a highly fermentative fluid.§

The absolute definitive surgical treatment for pseudocysts of the pancreas should therefore be the simplest and safest form of internal drainage available to the surgeon. Cystogastrostomy appears to be ideally suited to most cases of pseudocyst. Whether the accumulation of fluid be either in the lesser omental sac itself or actually subcapsular is of little importance in the eventual treatment. This is readily apparent when one realizes that the lesser sac is actually only a potential sac and really a peritoneal cleft and that nearly any inflammatory condition in the pancreas itself or in the posterior wall of the stomach effects a rapid adherence between the posterior wall of the stomach and the anterior peritonized surface of the pancreas. One can really appreciate this after encountering a pseudocyst or cystadenoma of the pancreas and seeing how, of all contiguous structures, it adheres to the posterior wall of the stomach, first and foremost. Some of the dangers of cystojejunostomy, performed either after the method of Roux or in a tangential manner, are due to the facts that the wall of the pancreatic cyst may be quite friable and not hold its sutures well and that there has been no peritoneal sealing about the anastomosis preoperatively. In

‡ Reference 34, pp. 199-200.

§ References 18 and 25.

performing a posterior cystogastrostomy, the very dense sealing of the peritoneum between the two viscera has long since occurred and all that remains to be done is to destroy a portion of the wall between the stomach and the cystic space to create a snug, sealed anastomosis with much less risk of peritoneal insult by cyst or enteric content. It is certainly not wise to dissect the posterior stomach wall away from the cyst and then create an anastomosis as has been recorded occasionally. The soundest procedure, as has been previously noted,‖ seems to be a transgastric cystogastrostomy in which the inflammatory sealing between the posterior wall of the stomach and pancreatic cyst is not disturbed.

Extragastric pancreaticocystogastrostomy has been done successfully.¶ However, the only recorded clinical failure of pancreaticocystogastrostomy for pseudocyst of the pancreas, which was presented in 1950 by Maxeiner and Maxeiner,[20] was an extragastric anastomosis along the greater curvature. Six days following this surgery, the patient commenced having multiple episodes of massive gastrointestinal hemorrhage. This patient underwent reexploration on the 45th postoperative day, and it was found at this time that the pancreatic cyst had practically completely disappeared, leaving only a scar on the greater curvature of the stomach. A gastrotomy done at that time revealed a posterior gastric ulcer adherent to the pancreas; this ulcer was then excised. In the immediate second postoperative period, the hemorrhages again recurred. Reoperation a third time, performed 20 days after the second procedure, was carried out to effect a subtotal gastrectomy in order to control the hemorrhage. At this operation, a peptic ulcer was described by the authors at the site of the old cystogastrostomy. A liver biopsy at this time showed acute hepatitis. In this patient, despite the fact that the cyst had obliterated itself and there was no patent opening between pancreatic tissue and the gastric lumen itself at the time of the second operation, recurrent peptic ulceration developed with the identical symptoms which were present prior to the second operation. Certainly a peptic ulcer on the posterior wall of the stomach of any size could have been present and remained undetected during the first operation, when a large amount of inflammatory reaction was already present in the pancreatic area due to the pseudocyst. Since an anterior gastrotomy was not done at the first operation and thus the interior of the stomach may not have been thoroughly explored, it is certainly possible that the ulcer was present at the time of the cystogastrostomy but was overlooked and that in the immediate postoperative period, as the cyst was evacuating itself, some of the pancreatic ferments may have lysed blood clots or thin blood vessel walls in the bed of the ulcer and precipitated the bouts of hemorrhage. This case, then, by no means can be recorded as a failure of cystogastrostomy to effect a cure of pseudocyst of the pancreas; it only emphasizes the necessity to establish the anastomosis by means of a transgastric approach (anterior gastrotomy).

One has only to review the number of cases in which pancreatic cysts have been successfully anastomosed to the stomach to discover the adaptability of the stomach to the modified pancreatic secretions contained in the cysts for the time necessary to allow the granulation-tissue-lined cyst to obliterate itself. The obliteration of the cyst has been confirmed not only by occasional subsequent reoperations but also by numerous reported follow-up gastrointestinal studies on these patients (as in both of the cases reported below).

‖ References 4, 30, and 26.

¶ References 8 and 15.

Further evidence to support the contention that the stomach can adapt without incident to pancreatic secretion is presented by Wells and his colleagues,[32] who reviewed the work of one of these authors (Wells) wherein successful pancreaticogastrostomies were done experimentally in dogs and who presented three pancreaticogastrostomies done in human patients after pancreaticoduodenectomy for carcinoma of the head of the pancreas. These authors stated that they believed pancreaticogastrostomy was safer than pancreaticojejunostomy and recorded all their operations as being technically successful. Dill-Russell [10] recorded two similar cases in 1952 in which the pancreatic neck was inserted into the stomach through the posterior gastric wall after pancreaticoduodenectomy for carcinoma of the head of the pancreas, again with no evidence of postoperative reaction in the stomach and with apparently successful postoperative anastomoses. Ingebrigtsen and Langfeldt [16] went a step further in 1952 and at the time of implantation of the pancreatic neck into the posterior wall of the stomach after pancreaticoduodenectomy for pancreatic cancer performed a concomitant antrectomy in the hope of postoperatively eliminating gastric hydrochloric acid, which might possibly destroy pancreatic trypsin. Postoperatively, an absence of free hydrochloric acid in the gastric secretions made it possible to assay gastric secretions for the presence of pancreatic enzymes. Ingebrigtsen and Langfeldt subjected two of their patients with carcinoma of the head of the pancreas to this procedure, and in both cases active pancreatic enzymes were found in the gastric contents postoperatively. Their conclusion was that the operations were technically and physiologically successful.

Here, then, is more evidence showing that the stomach can receive, with or without antrectomy, pancreatic enzymes from the duct of Wirsung itself with no apparent untoward reaction in the mucosa of the stomach. Certainly it seems logical to assume that the stomach can safely accept the usually dilute and temporary secretions of a pancreatic pseudocyst until the cystic space has obliterated itself through collapse and repletion by granulation tissue, which subsequently cicatrizes.

Thus it appears that pancreatic cystogastrostomy is an ideal form of treatment for pseudocysts of the pancreas in the majority of cases.

The only cases in which excision is believed to be best indicated are those few in which the cyst is quite small, readily mobilized from adjacent structures, and confined to either the body or the tail of the pancreas.

Presented below are two cases of pseudocyst of the pancreas, both of which apparently were secondary to pancreatitis as a result of biliary tract disease, and in each of which a curative transgastric pancreatic cystogastrostomy was done.

REPORT OF CASES

CASE 1.—E. S., a 51-year-old Negro was admitted to Baylor University Hospital, Sept. 6, 1951, with the complaint of gnawing epigastric pain of eight months' duration associated with nausea and vomiting. His family and past histories were noncontributory.

This patient had been completely well until eight months prior to admission, at which time he commenced having attacks of gnawing midepigastric pain with occasional sharp exacerbations radiating slightly to the right upper quadrant. These attacks, occurring every six to eight weeks, were accompanied by nausea and vomiting and were followed by residual epigastric and right hypochondrial tenderness and soreness for four or five days. He specifically stated that he had not had hematemesis, jaundice, change in bowel habits, or tarry or alcoholic stools. Considerable anorexia during the present illness had contributed to a 44 lb. (20 kg.) weight loss.

Physical Examination: This was essentially negative, except for the abdomen, which revealed a mass in the epigastrium approximately 22 cm. in diameter, which was hard, slightly tender and nodular and did not move with respirations. The mass was rigidly fixed.

Laboratory Studies: Stool examinations, urinalysis, all hematological data and blood serum studies were normal except for the serum bilirubin, which was 1.4 mg. per 100 cc., 1.0 mg. reacting directly. Gastric analysis revealed no free hydrochloric acid and 31 units of total acid. An upper gastrointestinal series revealed marked anterior displacement of the body and pylorus of the stomach by a large retrogastric mass with no evidence of an intrinsic gastric lesion. A chest x-ray revealed no evidence of disease except for a questionable area of plate-like atelectasis in the right lower lung field. Results of a barium enema were normal.

Course in Hospital: On admission to the hospital, temperature, pulse and respirations were normal, but on the third hospital day a sudden temperature elevation to 38.3 C. (100.9 F.) occurred and increased tenderness in the region of the abdominal mass was noted.

On Sept. 10, 1951, under endotracheal anesthesia, the abdomen was entered through a right upper transverse abdominal incision. A large amount of greenish turbid watery fluid was encountered in the peritoneal cavity. The gall bladder was found to be thickened, hyperemic, and edematous, and contained numerous small stones. A large grapefruit-sized mass was seen in the region of the pancreas with the stomach stretched over it anteriorly. The uppermost portion of the lesser curvature of the stomach was densely adherent to the undersurface of the left lobe of the liver. As the adhesions were broken down in this area, a subhepatic abscess was found herein where the pancreatic cyst had eroded through the gastrohepatic ligament and ruptured anteriorly, accounting, first for the moderate peritonitis, and, second, for the subhepatic abscess. In addition, a left subphrenic abscess, containing a few ounces of a milky, watery liquid, was found and evacuated by suction. Owing to the severe inflammatory reaction about the cyst, a circular posterior cystogastrostomy 3 cm. in diameter was made through an anterior gastrotomy incision at the junction of the middle and lower thirds of the stomach. The lesser bursa was found to be completely obliterated by the pericystic inflammatory reaction; 1,500 cc. of a creamy white fluid were aspirated from the cyst. The edges of the cystogastrostomy stoma were secured for hemostasis by means of a continuous, simple, locked No. 00 atraumatic absorbable (gut) suture, which was whipped about its entire circumference. Several specimens for biopsy were then taken from the interior of the cyst wall. The cyst lining appeared to consist of necrotic and granulation tissue; no evidence of epithelization or papillation was noted grossly. The tip of the intragastric Levin tube was placed through the cystogastrostomy opening into the cyst. The anterior gastrotomy wound was then closed with a running Connell-type suture of No. 00 chromic catgut followed by a reinforcing layer of seromuscular Lembert sutures of No. 30 black cotton. The perforation of the cyst through the gastrohepatic ligament was closed with interrupted No. 30 black cotton mattress sutures, this suture line being further reinforced with absorbable gelatin sponge (Gelfoam) and a patch of omentum. The common duct was aspirated at this point with a needle and found to contain clear watery bile; the gall bladder was then removed in routine fashion without any difficulty. The wound was closed in layers with black cotton suture, one Penrose drain being placed in Morrison's pouch and brought out through the lateral angle of the wound. The patient had withstood the procedure well and had received 500 cc. of blood intravenously.

Postoperatively, the patient was prescribed penicillin, 100,000 units every three hours, and dihydrostreptomycin, 0.5 gm. every six hours, both intramuscularly. Constant Wangensteen suction by Levin tube was also maintained for the first seven postoperative days. Over this seven-day period, he was given the necessary intravenous fluid replacements, including moderate amounts of calcium which aided in maintaining normal serum-calcium values postoperatively. On the eighth postoperative day, the Levin tube was removed and the patient was placed on a clear liquid diet, which he tolerated well. On the ninth postoperative day he was placed on a bland diet; he was discharged after a smooth convalescence on Sept. 20, 1951 (11th postoperative day). In the postoperative period, the average daily amount of bile-colored fluid obtained by suction was 500 cc.; the patient was afebrile postoperatively. The only pain of which the patient complained postoperatively was incisional pain. The drainage site at the lateral angle of the wound sealed completely on the eighth postoperative day, the drain having been removed on the fifth postoperative day.

Pathological Examination: Microscopic examination of the button of tissue excised in performing the cystogastrostomy and the fragments of cyst lining revealed no evidence whatever of any epithelial lining in the pancreatic cyst. Sections of the gall bladder wall were compatible with chronic cholecystitis and the gall bladder was found to contain many soft yellowish stones.

The patient was followed in the Baylor Surgical Clinic over the subsequent 12 months and was found to have been completely free of pain and to have resumed his normal activity in the eighth postoperative week, at which time he was placed on a general diet. The wound had healed completely per primam, and examination of the abdomen failed to reveal any evidence of the mass or of tenderness. At the end of 12 months, the patient had gained 27 lb. (12.2 kg.) in weight and was completely asymptomatic. An upper gastrointestinal series performed eight weeks postoperatively failed to reveal any regurgitation of barium into the cyst or gastric ulceration. Repeat upper gastrointestinal x-ray studies nine months postoperatively disclosed no evidence whatever of ulcerations, mass or internal sinus or fistula.

Case 2.—Mrs. T. B., a 64-year-old white woman, was admitted to Baylor University Hospital on Feb. 26, 1952, with a complaint of difficulty in swallowing, with occasional vomiting, of six weeks' duration. The family history was noncontributory.

Past History: This inquiry revealed only a hysterectomy with an anterior and posterior vaginal repair sustained at the age of 49 years.

Present Illness: Her current illness commenced approximately two years prior to admission, at which time, because of right upper quadrant pain radiating through to the interscapular area associated with nausea and vomiting, the patient was explored for gall bladder disease. At the time of exploration (at another hospital), a large retroperitoneal cyst was identified on which a biopsy was performed and found to be a pseudocyst of the pancreas; nothing further was reportedly done at that operation. The surgeon in attendance at that time stated he was unable to identify the gall bladder because of the mass. Since that time, the patient had gained weight and felt perfectly well until approximately six weeks ago, at which time she became aware of a sense of food sticking in her upper abdomen after a meal, associated occasionally with vomiting without nausea. During these six weeks she also became aware of a soft, nontender mass in her upper abdomen which she believed had increased somewhat in size over this period of time. During this same interval, she also noted occasional bouts of mild epigastric aching pain radiating through to the back. There was no history of jaundice.

Physical Examination: The abdomen revealed a well-healed right subcostal incision, a similar well-healed lower midline abdominal incision, and a bowling-ball-sized, firm, smooth mass occupying the upper midabdomen. This mass was rather fixed and nontender, moving only slightly with respirations; the percussion note over the mass was flat.

Laboratory Studies: Urinalysis, all hematological data, and serum chemical data were normal. Upper gastrointestinal x-rays revealed marked anterior displacement of the stomach and inferior displacement of the third and fourth portions of the duodenum. A gall-bladder series failed to demonstrate any function.

Course in Hospital: On Feb. 28, 1952, through an upper midline incision extending from the ensiform cartilage to the umbilicus, exploration of the abdomen was carried out. The gall-bladder area, the hepatic flexure of the colon, the pylorus of the stomach, and the first portion of the duodenum were markedly matted together by dense adhesions and compressed against the undersurface of the liver by the mass; the gall bladder was not visualized. The stomach was found to be tightly stretched anteriorly over a large fluctuant mass, which measured 25 cm. in diameter and arose from the region of the body of the pancreas. A cystogastrostomy was then established exactly as in Case 1, and 2 liters of green, turbid watery fluid was aspirated from the cyst. A very dense inflammatory sealing was noted between the posterior serosa of the stomach and the cyst wall in this case also, the lesser bursa being obliterated. Examination of the interior of the cyst revealed only granulation tissue in the wall, with no gross evidence of neoplasia. The anterior gastrotomy wound was then closed, after which the operative wound was closed without drainage.

Postoperatively, the patient was placed on a regimen of penicillin, 400,000 units intramuscularly twice daily, and constant Wangensteen suction through a Levin tube. On the fourth postoperative day, the Levin tube was removed and hourly feedings of small amounts of milk and water were commenced. Over the subsequent five days this diet was increased to a regular bland ulcer diet, the patient tolerating it extremely well. On the fourth day methantheline

(Banthine) bromide orally was prescribed, 50 mg. four times a day in an attempt to suppress both gastric and pancreatic secretions temporarily. Her convalescence was completely uneventful, and she was discharged from the hospital March 8, 1952 (the tenth postoperative day).

Pathological Examination: Microscopic examination of the button of tissue removed revealed stomach wall in which there was a moderate chronic gastritis and cyst wall comprised of dense fibrous connective tissue with a superficial layer of loose, highly vascular granulation-type tissue; there was no epithelial element in the cyst wall.

Since her discharge from the hospital the patient has noted no recurrence whatsoever of vomiting, abdominal pain, dysphagia, or the mass. Reexamination eight months postoperatively revealed a normal abdomen to inspection and palpation except for the three well-healed incisions. An upper gastrointestinal series at this time failed to reveal any abnormality and specifically showed no evidence of any internal sinus or fistula.

SUMMARY

Argument is presented as to why transgastric pancreatic cystogastrostomy is believed to be the safest and most logical definitive therapy for pseudocyst of the pancreas. The simplicity of the operation itself and the extremely smooth postoperative convalescence following this operation are stressed.

Clinical features of pancreatic pseudocyst are briefly described. The interesting history of the surgical attack upon pseudocyst is reviewed.

Two additional cases are added to the surprisingly small group of known transgastric pancreatic cystogastrostomies.

The frequently severe morbidity, technical difficulties, relatively high mortality, and recurrences associated with the remaining means of treatment of pancreatic pseudocysts (marsupialization, cystovisceral anastomosis other than cystogastrostomy, and excision of the cyst with or without partial pancreatectomy) are briefly presented.

It is reiterated again that the only ideal therapy for all proliferative (neoplastic) cysts of the pancreas is total extirpation.

Dr. Sam. R. Burnett, Dallas, Texas, gave permission to include his case (Case 2) in this report.

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TRENDS IN ORTHOPEDIC SURGERY

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LOS ANGELES

THE SCOPE of modern orthopedics is both medical and surgical. It is a field that is being cultivated with the newest implements of basic science research. Advances in biological research are being applied to orthopedic surgery now more than ever before in history. Papers in orthopedic surgery during the past few years have dealt with embryonic life and the whole span of childhood, maturity, and senility. Newer knowledge has been sought even in ancient bones and anthropological specimens.[1]

Disorders and treatments regarded as purely mechanical in the first half of this century are now more accurately described as biomechanical and are being investigated with the aid of the methods of basic chemistry and physiology of living processes. The Josiah Macy Jr. Foundation made an outstanding contribution in organizing multidisciplinary conferences to encourage communication and integration of biological researches on bone and connective tissue. The Conferences on Metabolic Interrelations from 1949 to 1953 and on Connective Tissue from 1950 to 1955 have outlined the major advances and problems in the physiology of bone and joints. The subjects were discussed on both clinical and experimental levels. A similar conference to advance the study of structure, composition, and growth of mineralized tissues was sponsored by the New York Academy of Science on Jan. 22 to 23, 1954, to include research done on bone and teeth. From July 10 to 23, 1954, a Gordon Research Conference on the chemistry, physiology, and structure of bone was sponsored by the American Association for the Advancement of Science, for 100 investigators from all over the world, and assembled at Kimball Union Academy, Meriden, N. H.

Widespread enthusiasm for fundamental research in orthopedic surgery demanded serious attention and received prompt action in 1953. According to survey work by A. R. Shands Jr.,* who polled 2,250 members of the American Academy of Orthopedic Surgeons, the lack of facilities and space, not simply lack of funds, was found to be the principal deterrent to research. The total sum of $430,000 was being expended yearly. Most of these funds were in the form of 19 large grants totaling $340,000 from federal agencies, private foundations and major universities. Shands organized a committee to formulate plans for an Orthopedic Research Society and an Orthopedic Research Foundation to be established by the American Academy of Orthopedic Surgeons. The Foundation is to be a nonprofit organization supported by funds from various sources to expand present facilities and areas and to encourage orthopedic surgeons who have the desire to do basic experimental and clinical research.

Department of Surgery, University of California School of Medicine at Los Angeles (Dr. Urist).

* Shands, A. R., Jr.: Personal communication to the author.

THE STRUCTURE AND CHEMICAL COMPOSITION OF BONE MATRIX

The need for intensive investigation of the organic phase of bone has been made very obvious by the limitations of our present knowledge of the inorganic phase. Electron microscopy and combined histological, chemical, and histochemical methods have shown that the organic matrix has two components: collagen and a homogeneous material. The homogeneous material is a fluid resembling fluid of other tissues and amorphous ground substance identifiable as mucopolysaccharide. The collagen in bone appears morphologically to be the same as collagen in connective tissue everywhere in the body. It has a fibrillar appearance with double crossbanding at intervals averaging 640 Angstrom units (A.). The ground substance of bone, like other connective tissues, produces characteristic staining reactions: metachromasia with toluidin blue and a positive reaction with the Hotchkiss procedure. The latter has the distinction among histological staining methods of being a true chemical reaction. It depends upon the oxidation of hydroxyl or hydroxyl-amino groups in carbohydrate molecules to aldehyde groups. It has been suggested that depolymerization makes hydroxyl groups available for the Hotchkiss reaction and gives a more intense pink stain. Polymerization gives a slight or no positive reaction. How polymerization and depolymerization of ground substance is involved in formation and calcification of bone poses a question of extreme practical importance to all those who deal with the skeleton in health and disease.†

THE MINERAL PHASE OF BONE

More is now known about the physical structure of the bone salt than of its exact chemical composition. Electron microscopy reveals that the crystals of bone are minute hexagonal shaped tablets, approximately 400 A. long (with the "C" axes of the unit cells oriented in its long dimension), 250 A. in width, and 25 to 50 A. in thickness. X-ray diffraction studies supply proof of the accuracy of these measurements and show that they are more than interesting approximations. Slightly different dimensions have been recorded, but the differences between estimates are extremely small. One crystal may be slightly smaller or larger than another. What is absolutely invariable is the geometric configuration. The crystal is formed in the shape of hexagons and parallelapipeds whose angles are constant, determined by energy relationships between atoms of calcium and phosphorus and the arrangements of these elements in space.

The crystal is closely associated with collagen lying between the characteristic bandings of the fiber. It is oriented in the long crystal axis (and the c-axis) parallel to the longitudinal direction of each fiber. The crystal consists of calcium, phosphate, and hydroxyl ions which defract x-rays to give a pattern characteristic of apatite minerals. The surface area of the bone mineral because of the minute size of the crystals, is enormous, measuring at least 80 acres in a man weighing 160 pounds. Surface phenomena dominate the chemical behavior of the bone salt. The most important process, recently demonstrated, is ionic exchange with the medium bathing the surfaces of the crystals. Deposition of radioisotopes gives complete confirmation of previous chemical observations of the dynamic equilibrium between blood and bone. In addition, studies with isotopes have shown striking variations in reactivity

† References 2 and 3.

from bone to bone, in metaphysis as compared with diaphysis, and in new as compared with old layers within a single haversian system. The surfaces actually become less and less reactive with increasing age of the crystal. Radioactive isotopes are removed from the extracellular fluid and deposited in bone by (1) surface exchange with ions in mineral crystals, or (2) a specific but as yet uncharacterized deposition in organic matrix.‡

RADIOISOTOPES IN BONE METABOLISM

The irradiation effect is the chief characteristic which determines the usefulness of any one radioisotope in experimental and clinical orthopedics. Isotopes with a half-life of less than 14 hours are practically useless, while those of a half-life of over 30 to 40 days yield exceedingly dangerous radiation levels. Thus human application has been restricted to some extent by these limiting specifications. A considerable number of isotopes are, however, available for clinical investigations. Several isotopes, such as those of gallium, indium, and strontium, can localize in osteogenic sarcomas; P^{32} accumulates in malignant cells more than it does in normal cells. Unfortunately, even with the capacity to concentrate radiation at the critical point with maximum intensity, none of these isotopes have proved a satisfactory treatment for osteogenic sarcoma. The histologic changes that follow beta and gamma radiation from the use of radioactive isotopes seem comparable to those seen following multiple lower intensity doses of x-radiation and fast neutrons. Further experimental research with metabolic vehicles for radioactive isotopes should greatly advance knowledge about bone in health and disease.[6]

METABOLIC BALANCE STUDIES

There are several important contributions to our knowledge of the physiology of bone in man which were made possible by metabolic studies and which could not have been made by any other method. The minimum daily requirement of calcium and phosphorus for infants, children, and women during pregnancy and lactation are well known. Metabolic studies have revealed the negative calcium balance in patients with hyperparathyroidism and kidney stones in stages and forms of the disease in which there are no bone changes demonstrable by x-ray. Balance studies are also extremely important in diagnosis of rarer conditions like the Fanconi syndrome, situations in which ordinary renal function tests are normal but the patient is binding organic acids with the body reserves of fixed base. Bone calcium is a large source of fixed base which the skeleton does release on demand. A new question is the role of sodium in the metabolism of bone. Apparently, a large amount of calcium and phosphorus may be mobilized from the skeleton to remove a relatively small amount of sodium. The biochemical and endocrine forces are not yet known.[7]

Balance studies are particularly useful in clinical investigations where it is undesirable to wait months or years of valuable time for gross improvement to determine whether therapy is effective. One example is the discovery that a mixture of citric acid and sodium citrate will spare serious loss of calcium in patients with

‡ References 4 and 5.

the hyperchloremic type of acidosis. Another is that estrogen treatment will produce retention of nitrogen, calcium, and phosphorus in patients with senile osteoporosis before any bone changes are demonstrable in roentgenograms.

TISSUE CULTURE

Fell's [8] method of culture of bone anlagen permits study of the response of isolated skeletal tissues to products of intermediary metabolism, vitamins, and hormones. Laboratory studies along these lines, although just beginning, show that a vitamin can affect the tissue without being mediated by the whole animal. Recent work with vitamin A is an excellent example of a substance that is active in vitro in concentrations in which it occurs in vivo. With 3,000 I. U., in tissue culture, the amount that can be obtained in the blood stream in vivo, there is interference with the metabolic processes which form cartilage matrix. The intercellular substance loses its metachromatic staining and rapidly disappears, while the chondrocytes continue to proliferate. Vitamin A produces these effects only through cellular activity. The cartilage cells fail to synthesize condroitin sulfate in medium saturated with vitamin A and will lose what matrix they had previously accumulated. Cartilage models heated to 45 C., which just prevents growth, cannot produce these effects of vitamin A. The animal may do something to the vitamin in vivo by forming a protein complex to store it and control the active amount of substance.[8]

HORMONES

The interrelationships between the endocrine and skeletal systems are many and complex. Parathyroid hormone and somatotropic hormone of the pituitary gland in particular have effects which are specific for the skeleton. Estrogen in certain species of animals, notably birds and mice, also has a special effect upon bone tissue. Well-controlled experimental work indicates that the theory that parathyroid hormone has only secondary effects upon bone is probably incorrect. Parathyroid gland transplants to a host bed of bone produced marked and specific local resorption of bone tissue.[9] Growth, or somatotropic, hormone has been prepared in pure form and is capable of sustaining the growth apparatus of a hypophysectomized animal.[10] Large deposits of endosteal new bone can be produced within a few weeks in the bones of estrogen-treated mice, but no similar effect has been elicited in the human species. Experiments with radioactive estrogen 16-C^{13} show selective concentration of radioactivity in bone and extramedullary hematopoietic tissues.[11] When treated with large doses of estrogen, patients with osteoporosis show relief of pain and a prolonged positive calcium balance. X-ray evidence of increase in bone density has not been satisfactorily demonstrated. At least 30% change in bone substance is necessary before it is demonstrable in roentgenograms, and many years of treatment may be necessary before the bones are appreciably increased in density.

HEALING OF FRACTURES

The significance of cartilage and fibrocartilaginous callus in fracture healing has been determined from the end-results of transplants to the anterior chamber of the eye [12] and the capsule of the kidney.[13] In fractures with displacement or large defects, cartilage functions as an inductor which draws osteogenesis into and across the fracture gap. In some unknown way, it also organizes the skeletal tissues in the normal order that is essential for bone regeneration.

BONE BANKS

Fresh autogenous bone is the most commonly available and satisfactory material for bone graft operations. Homogenous bone, heterogenous bone, and even autoclaved bone, however, have been used successfully in every form of bone graft operation and continue to be used in clinics all over the world.[14] Urist reviewed the experimental and clinical literature [15] and concluded that the success of an operation is determined, first, by the proliferative activity of the host bed and, second, by the donor tissue. The theory of induction offered a reasonable hypothesis for the further study of replacement of a bone graft. Induction, the physical-chemical effect of one tissue upon another in contact with it, formulates the best explanation for successful results with autoclaved bone.[16] It is important to realize that bridging of a defect with a massive bone graft is frequently unsuccessful and requires many years of healing, even with fresh autogenous bone. It is especially risky with autoclaved bone, which must have a perfectly prepared host bed and the maximum surface contact. Complications with bank bone,[17] such as transmission of tuberculosis from ribs collected at thoracoplasty operations and homologous serum jaundice from iliac bone [18] of random donors, indicate that bank operators should take every possible precaution in accepting deposits of bone tissue.

SUMMARY

This brief review indicates the importance, the need and the many opportunities for a greater understanding of bone as a tissue. The fallacy of treating bones as mechanical objects is now well known to the great majority of those who practice orthopedic surgery. As facilities and funds for basic research in both academic and service hospitals are enlarged and broadened to include divisions of orthopedics, there is also likely to be greater evidence of interest in basic science by orthopedic surgeons. Like any other surgical specialty, orthopedics deals with clinical problems on the level of gross anatomy. Like other fields of medicine, its scope grows with new methods in basic science and its progress is made by advances made not only by those within its own discipline but very often by others working in other disciplines. Before Lister did very many successful operations on human viscera he made a major contribution to orthopedic surgery by applying his method of asepsis to the problem of repair of injuries of the patella. Lister was one of the first to see the application of Pasteur's microbiological researches to modern surgery. This is one of history's many examples of how he who labors with basic research, as an endeavor in its own right, can produce either the things which grow in his own field—or seed which can be blown over and grown in fields of others.

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TRENDS IN ORTHOPEDIC SURGERY

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ORTHOPEDIC SURGERY IN CEREBRAL PALSY

A RECENT but definite trend in the orthopedic treatment of the cerebral palsied has been clearly and concisely stated by Baker.[1]

Twenty years ago the therapeutic pendulum had swung to the extreme end of the operative scale, and orthopedic teachings were replete with various procedures to lengthen tendons, capsulotomies, and total and subtotal neurectomies of peripheral nerves. Approximately 10 years ago the pendulum swung to the opposite extreme and operative treatment was deplored except as a final resort in the correction of deformities which had been treated by manipulative means over a long period of time with no improvement. Generally speaking, orthopedic surgery was considered to be contraindicated in cases with athetosis or tremor, rarely indicated in cases with ataxia, and seldom needed in the spastic or rigidity types of cerebral palsy.

The therapeutic pendulum has now swung away from these two extremes and is occupying a more nearly midposition. Specific deformities in the cerebral palsied interfere with motor development and the acquisition of specific skills necessary in the performance of daily activities. These deformities should be analyzed carefully as to their cause, location, and extent. The agonistic and antagonistic muscle groups must be carefully evaluated. With use of this information, an attempt should be made to correct the specific deformity before instituting the cerebral-palsy treatment program. Mechanical devices such as wedging plaster casts or braces may be given a preliminary trial in the mild or moderately severe deformities. If this deformity does not respond readily, it should be corrected surgically. After the deformity has been corrected, intensive habilitative measures may then be indicated as necessary in the particular case.

Baker [1] has described a method of correcting equinus deformities of the ankle by detachment of the tendons of origin of the gastrocnemius muscle from the lower femur. This procedure is indicated in patients with equinus deformity of the ankle which is caused by a contracture or spasticity of the gastrocnemius muscle without significant involvement of the soleus muscle.

USE OF ISOTOPES IN ORTHOPEDIC SURGERY

Up to now radioactive elements have proved to be of little value from a therapeutic standpoint, but they may ultimately prove to be of very great value in scientific investigation of the metabolism of bone and cartilage.

As has been often repeated, the treatment of fractures of the neck of the femur is still an unsolved problem. The two complications most feared in such cases have been nonunion and avascular necrosis of the head of the femur. With the use of

adequate roentgenographic control and adequate internal fixation, the incidence of nonunion has been significantly lowered. Avascular necrosis, which occurs in roughly one-third of the cases, is believed to be caused by interruption of the blood supply to the femoral head at the time of injury or treatment. The development of adequate methods for internal fixation of this fracture has not significantly lessened the problem of avascular necrosis as a complication. If the viability of the femoral head could be accurately established at the time of the initial fracture, treatment could be modified to prevent pain and disability and to eliminate the need for secondary reconstructive operations on the hip.

In 1950 Tucker [2] issued a preliminary report on the use of radioactive phosphorus (P^{32}) to determine the circulatory status of the femoral head complicating fractures of the neck of the femur. Radioactive phosphorus was injected intravenously, and two hours later, when the fracture was reduced, biopsies of the femoral head and of the greater trochanter were done. The specimens were washed, dried, ashed, and assayed with a Geiger counter, and a ratio was established between the counts in the trochanter and the counts in the femoral head. A low ratio between trochanter and head was regarded as indicating a good prognosis, and a high ratio was considered of poor prognosis in regard to the viability of the femoral head.

Boyd, Zilversmit, and Calandruccio * began investigating the same problem in 1949 and have used a different technique. Radioactive phosphorus is injected intravenously, and two hours later, at the time of reduction of the fracture, a specially constructed Geiger counter is inserted into the trochanter and counts are made. The counter is then inserted into the femoral head through the guide-wire channel, and readings are made. A trochanteric to head ratio is thus established. Data have been obtained from approximately 75 patients in an attempt to establish normal ratios and ratios in cases with fracture of the neck of the femur. The latter cases must be followed closely over a long period of time to establish the incidence of avascular necrosis in these particular instances.

From the above it is apparent that with the use of new investigative methods, orthopedic surgeons are making progress in the solution of an old problem, namely avascular necrosis of the head of the femur complicating fracture of the neck of the femur.

ETIOLOGY AND TREATMENT OF LETTERER-SIWE DISEASE

Fisher [3] recently published an interesting report which at least partially clarified some of the confusion existing in regard to the diagnosis and treatment of certain of the histiocytic diseases of bone.

It is a recognized fact that neither roentgenographic nor histologic examination can always permit a clear distinction between eosinophilic granuloma of bone and the skeletal lesions associated with either Hand-Schüller-Christian disease or Letterer-Siwe disease. Rather, the three conditions must be distinguished by the clinical course of the affected patient. The clinical manifestations in a given patient are considered to be the result of an interplay between certain resistance factors in the host and the virulence and dissemination of the offending organism.

* Boyd, H. B.; Zilversmit, D. B., and Calandruccio, R. A.: The Use of Radioactive Phosphorus (P^{32}) to Determine the Viability of the Head of the Femur, personal communication to the author.

Fisher reported a case of Letterer-Siwe disease apparently caused by a paracolon bacillus of the Arizona group, which, after a short and near-fatal course, responded to antibiotic treatment, chloramphenicol (Chloromycetin) and oxytetracycline (Terramycin) being used. There are at least four other known cases of Letterer-Siwe disease which have responded favorably to antibiotic therapy.

Fisher [4] recommends that careful bacteriological examinations be made in suspected cases and especially for the paracolon bacilli. Even in the absence of a positive culture, he advises a trial of chloramphenicol and oxytetracycline in similar cases.

PROGRESS REPORT ON THE AMERICAN SURGICAL MATERIALS ASSOCIATION

The Joint Committee to investigate the advisability of establishing an association to standardize surgical materials has rendered an exhaustive report recommending the establishment of such an organization and outlining its purposes, plans, and administration. This committee, headed by Dr. Joseph S. Barr, was appointed by the American Medical Association, the American College of Surgeons, and the American Academy of Orthopaedic Surgeons. The American College of Surgeons and the American Academy of Orthopaedic Surgeons have approved the Committee's report and have appropriated funds to support the American Surgical Materials Association (A. S. M. A.). The American Medical Association is withholding its support until certain legal questions can be cleared up. Their ultimate approval is highly desired and is expected.

The purpose of this organization will be research, development of standards, testing, evaluation of products, and provision of an information service, all in the field of surgical materials. After the organization begins to function, it will adopt an appropriate seal, the use of which will be permitted in marketing of accepted products.

The American Surgical Materials Association will lean heavily on the three above-mentioned parent medical organizations during the initial years of its development. In time, however, financial support is anticipated from the American Hospital Association, from insurance companies, and from manufacturers and fabricators of metal devices, plastic devices, and surgical sutures.

The development of this organization will satisfy a long-standing need by the elimination of mechanical failure of implants into the human body.

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Case Reports

RETROPERITONEAL METASTATIC DISEASE DUE TO CARCINOMA OF THE PROSTATE

Value of Serum Prostatic Acid Phosphatase in Identifying Retroperitoneal Metastasis Originating from Carcinoma of Prostate

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THIS IS an abstract of the pertinent data of a case in which there was an unusual spread of a prostatic carcinoma that regressed after orchiectomy and estrogen therapy. The case demonstrates the diagnostic possibilities of the sensitive method used for measuring serum "prostatic" acid phosphatase,[1] which showed that the "prostatic" fraction of the enzyme may be elevated to an abnormal level even when the total serum acid phosphatase, as measured by the customary King-Armstrong technique, is within the normal range.

REPORT OF A CASE

A 76-year-old man had for many years had frequency, which in the recent months prior to admission, had increased and been accompanied by extreme urgency. Five days prior to admission, acute, severe, lower abdominal pain with prostration developed. The patient was seen by his family physician. Abdominal tenderness associated with some spasticity was elicited, and an enlarged prostate that was suspiciously nodular was palpated. The acute symptoms were relieved by antibiotics, and the patient was transferred to the care of an internist and a urologist. At urologic examination a large hard mass extending from the pelvis to the level of the umbilicus could be felt. The prostate seemed nodular, but continuity with the abdominal mass could not be established. Well after the inflammatory process had subsided, a barium enema (Fig. 1*A*) and intravenous pyelography demonstrated a persistent retroperitoneal mass displacing the sigmoid to the left and fixing it in position.

The serum acid phosphatase level was well within normal limits at 4.0 King-Armstrong units per 100 ml. The serum "prostatic" acid phosphatase, as determined by Fishman and Lerner * in their laboratory, was definitely in the abnormal range at 2.9 units per 100 ml., the normal value for this determination being less than 0.5 units per 100 ml.

A series of x-ray films was negative for metastases. A transurethral biopsy of the prostate revealed an anaplastic adenocarcinoma of the prostate. When the patient was examined under anesthesia, the prostate was found to be contiguous with the abdominal mass. Orchiectomy was performed, and the patient was given 5 mg. of diethylstilbestrol daily. Within 10 days, the mass became noticeably smaller.

In the follow-up examination of the patient six weeks after his discharge from the hospital, the mass could no longer be felt and the serum "prostatic" acid phosphatase had decreased to a normal value (Fig. 2). Three weeks after the follow-up examination of the patient, a barium

W. H. Fishman, Ph.D., gave help and advice in supervising the laboratory work.

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This study was supported by a control project grant from the National Cancer Institute, National Institutes of Health, United States Public Health Service.

* References 1 and 2.

Fig. 1.—Barium enema (*A*) before, and (*B*) nine weeks after orchiectomy and diethylstilbestrol therapy for prostatic adenocarcinoma. *A* shows narrowing and poor filling of lower sigmoid (this area was immovable during fluoroscopy); *B*, disappearance of narrow area and return of normal filling in lower sigmoid area, freely movable on fluoroscopy at this time.

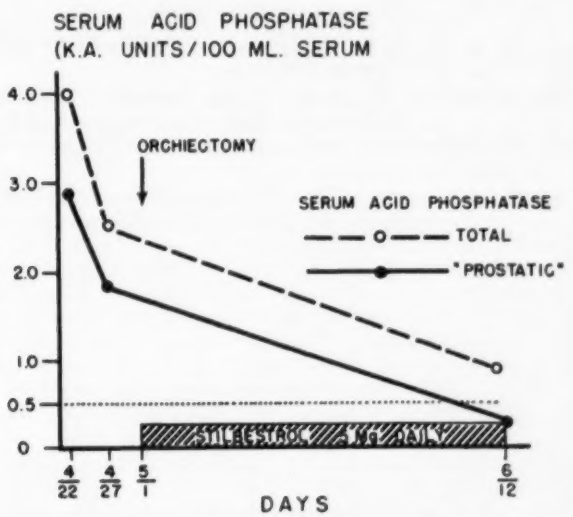

Fig. 2.—Determinations of "prostatic" serum acid phosphatase from April 22 to June 12. The return to normal of the enzyme after definitive therapy for anaplastic adenocarcinoma of the prostate (May 1) paralleled the clinical improvement.

enema confirmed the disappearance of the mass; and at fluoroscopy, the lower sigmoid was freely movable (Fig. 1*B*). Clinically, the patient is now symptom-free and carrying on his normal activities.

COMMENT

Carcinoma of the prostate gland may metastasize in any or all of three ways—by bloodstream invasion, lymphatic spread, or direct extension. Metastases via the bloodstream are frequently seen and appear most commonly in the lumbosacral spine and in the bones of the pelvis. The liver and lungs may also be involved. Lymphatic spread may be to pelvic, lumbar, thoracic, supraclavicular, femoral, inguinal, and at times, mediastinal nodes. Direct extension by contiguity causes invasion of the seminal vesicles, urethra, bladder, rectum, periureteral tissues, and pelvic fascia.

It is extremely rare for a metastatic lesion of the retroperitoneal area to become so large in size that the presence of the disease is demonstrated by a palpable abdominal mass extending to the level of the umbilicus. Only one instance of this striking manifestation has been reported in the recent literature.[3] It is felt that the abnormal value for the serum "prostatic" acid phosphatase determined by the method of Fishman and Lerner [1] aided materially in establishing the diagnosis of the case, which presented such an unusual sign.

SUMMARY

A case of carcinoma of the prostate presented a retroperitoneal metastatic mass of unusual size.

Although the total serum acid phosphatase (King-Armstrong units) was within normal limits, the serum "prostatic" acid phosphatase, as measured by the Fishman-Lerner technique, was definitely elevated and proved to be of diagnostic value.

Rapid regression of the mass followed orchiectomy and estrogen therapy.

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STRANGULATED DIAPHRAGMATIC HERNIA WITH GANGRENE OF STOMACH

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STRANGULATION of a diaphragmatic hernia is sufficiently rare an occurrence to warrant reporting in the literature. It is important that this diagnosis be kept in mind in any case of obscure lower thoracic or upper abdominal pain because the history and clinical picture of strangulated diaphragmatic hernia make an almost classic entity. Early diagnosis is essential for adequate therapy, whereas procrastination spells disaster for the patient.

Carter and Giuseffi [1] surveyed the literature in 1948 and collected 43 cases of strangulated diaphragmatic hernia. To these Pearson [3] later added 29 more from the literature and 4 of his own, to bring the total to 76 cases to date.

As far as involvement of the stomach is concerned, we have been able to find six reports of gangrene of the stomach due to strangulated diaphragmatic hernia. Hamilton and Phillips [2] in 1949 treated two cases of their own and collected three more from the literature. Subsequently, Pearson treated a case of gangrene of the entire stomach with the exception of 2 cm. of the pylorus. Our presentation is thus the seventh reported case.

REPORT OF A CASE

The patient, a 21-year-old white woman, was hospitalized at the Jewish Sanitarium and Hospital for Chronic Diseases on two occasions, June 15 to July 12, 1943, and Aug. 22, 1944, to Jan. 7, 1951. She suffered from cerebral palsy (Little's disease), and the care she received was primarily custodial.

On May 6, 1946, a routine chest film disclosed the following: "Lung fields are perfectly clear. The heart is in the midline. The right diaphragmatic leaf is well outlined. A dense band is seen across the left lower chest and several other bands are seen below this density. The appearance is that of gas-filled loops of bowel (Fig. 1).

"After administration of barium, one can demonstrate the greater portion of the stomach to be lying within the left thoracic cavity. The esophagus enters the stomach eccentrically above the diaphragm. The stomach is completely rotated. The greater curvature is turned upward and the lesser curvature faces downwards. About four-fifths of the stomach appears to be within the thorax. The duodenal bulb can be seen within the abdomen (Figs. 2 and 3). I believe we are dealing with a large hiatus hernia."

Barium enema showed the colon in normal position.

During the patient's course in the hospital prior to her final illness, two episodes occurred that involved the gastrointestinal tract. On May 16, 1948, she complained of abdominal pain and vomited dark-brown fluid. This was attributed to food poisoning, but the administration of morphine was necessary before relief was obtained.

From the Jewish Sanitarium and Hospital for Chronic Diseases.

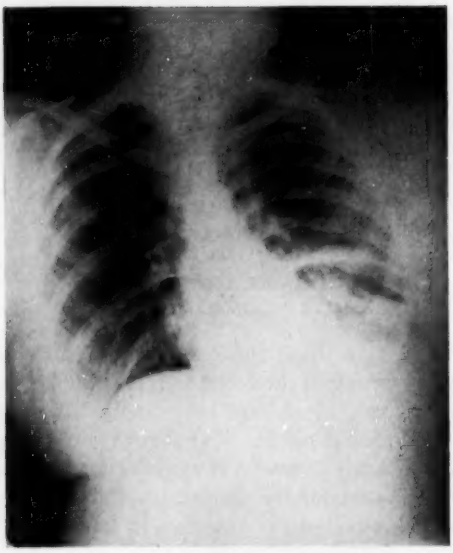

Fig. 1.—Routine chest film, May 6, 1946, suggesting the presence of bowel in the left chest (not to be mistaken for a high diaphragm).

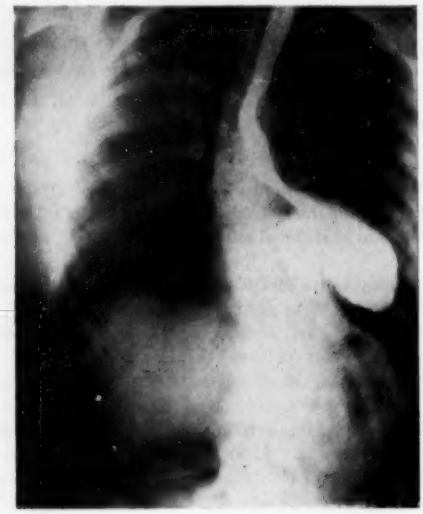

Fig. 2.—A large portion of stomach almost up to the antrum is seen above the diaphragm in the left chest.

Fig. 3.—The stomach has herniated through a large hiatus opening. The esophagus is seen entering eccentrically into the herniated portion of the stomach; the duodenal bulb is below the diaphragm.

Fig. 4.—Bedside film taken ante mortem. The trachea and heart are displaced to the right, and the left diaphragm is completely obscured. The haze covering the left lung field suggests either a pleural effusion or a fluid-filled stomach.

Again on Aug. 8, 1950, the patient complained of pain in the left upper quadrant. This was constant, nonradiating, and associated with nausea and gagging but no emesis. Momentary relief followed belching, but the pain recurred. There was no diarrhea and no elevated temperature. The attack gradually subsided under symptomatic therapy.

Her last illness began Jan. 6, 1952, when the patient complained of severe pains in the left upper abdomen and left lower ribs which, as previously, persisted despite sedation. She was nauseated but did not vomit. The findings were the same as on her previous attack. No friction rub was heard in the chest. Symptomatic therapy produced only transient relief.

By the next morning, the condition of the patient had considerately deteriorated. She was cyanotic and still complaining of the same pain. Her pulse was very thready and she was in apparent shock. Some improvement was obtained by the use of caffeine, nikethamide (Coramine),

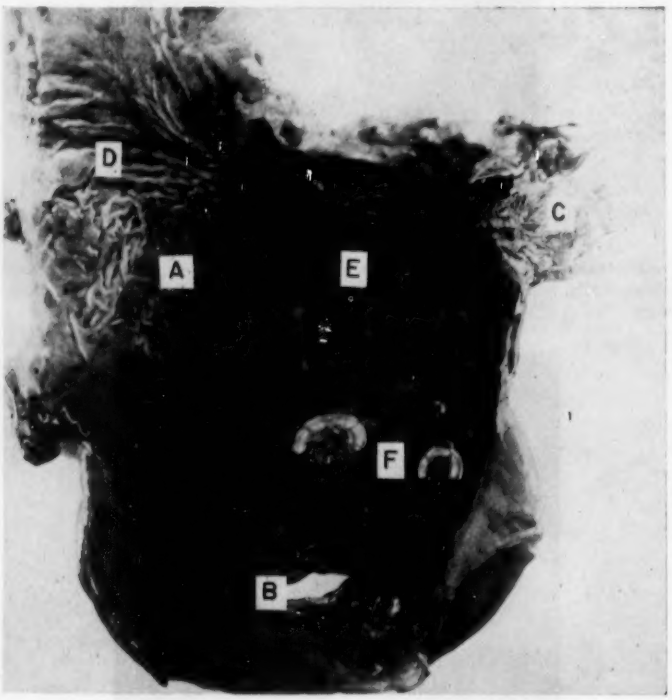

Fig. 5.—Postmortem specimen of stomach and esophagus: *A*, line of demarcation between normal and gangrenous stomach; *B*, perforation near greater curvature; *C*, and *D*, normal esophagus and stomach; *E*, gangrenous stomach; *F*, ulcerations in mucosa of stomach.

and oxygen, the blood pressure reaching 100/65. Bedside chest film was taken at this point (Fig. 4). Dullness and rales were present over the left chest front and back. Levin tube suction yielded some brownish fluid from the stomach, but the procedure exhausted the patient. She was coughing and complaining of shortness of breath. The pain in the left chest continued. Her breathing grew labored, and despite treatment she became progressively worse and died at 11:20 a. m.

POSTMORTEM FINDINGS*

The relevant postmortem findings were limited to chest and abdomen. The abdominal cavity contained about 500 cc. of a greenish-black semiliquid material. The upper half of the stomach

* Courtesy of Dr. B. W. Volk, Pathologist of the Jewish Sanitarium and Hospital for Chronic Diseases.

penetrated through the esophageal hiatus into the left pleural cavity. When the stomach was retracted into the abdominal cavity, a large gastric perforation about the size of a quarter was noted.

Both diaphragms were at the level of the fifth intercostal space. The left pleural cavity contained about 200 cc. of a fluid similar to that seen in the abdomen. The right pleural cavity was free of fluid. The plurae were smooth and shiny. The pericardial sac contained 25 cc. of a straw-colored clear fluid.

All lobes of the lung were crepitant. On sectioning they were dark-purplish and wet, and on pressure an abundant amount of frothy fluid escaped. The trachea and bronchi were lined by pale smooth mucosa.

Gastrointestinal Tract.—The esophagus was normal. The stomach was markedly distended in the oral portion. The wall was thin, soft, and friable. In this area the mucosa was dirty-gray, soft, and gangrenous. In the midportion there was a definite, sharp line of demarcation separating the proximal portion from the distal portion of the stomach. In the proximal portion the folds were flattened. A large perforation about 1.5 cm. in diameter was present near the junction of the two portions. The distal mucosa was pinkish and of normal appearance (Fig. 5). The remaining gastrointestinal tract showed no abnormalities.

MICROSCOPIC FINDINGS

Lungs.—There was marked congestion of the smaller and larger branches of the pulmonary artery. No other changes were noted.

Stomach.—Sections from the proximal portion showed extensive degeneration of the wall with disintegration of all layers without evidence of inflammatory reaction. Sections from the distal portion of the stomach showed no conspicuous abnormalities.

ANATOMICAL DIAGNOSES

The anatomical diagnoses were as follows: (1) incarcerated diaphragmatic hernia; (2) perforation of the greater curvature of the stomach into the left pleural cavity and abdomen; (3) congenital malformation of brain (cerebral palsy); (4) pulmonary edema and congestion; (5) passive congestion of the viscera.

COMMENT

As pointed out by Carter and Giuseffi,[1] in 85% of strangulated diaphragmatic hernia cases the pain is sudden in onset, usually related to an increase in intra-abdominal pressure, and in 88% of cases it is accompanied by vomiting either simultaneously or soon afterward; vomiting is only occasionally noted as an initial symptom. The pain is in the left upper quadrant or lower left thorax in the majority of cases, although it has been reported as epigastric, right lower quadrant, periumbilical, or generalized. Usually it is severe and colicky in type. Strangely enough, radiation into the left shoulder is not very frequent. Hiccup may be present as well as dyspnea. Constipation may likewise be present if the colon is in the hernial contents. The reporting of an effectual bowel movement and normal peristalsis may indicate involvement of the stomach only. Frequently the antecedent presence of constipation with straining at stool may precipitate an attack.

There is a history of diaphragmatic hernia in almost all cases, the traumatic variety (due to stab or gunshot wounds or indirect violence) being responsible for 79% of the cases in the series reported by Carter and Giuseffi, the rest being divided between congenital and indeterminate classifications.

The physical findings come next in elucidating the clinical picture. First and foremost, the abdominal findings are not of diagnostic consequence. There may be some tenderness in the left upper quadrant and possibly a concavity in the left

hypochondrium by reason of the migration of abdominal organs into the chest. But otherwise there is little in the abdomen to indicate a pathologic condition. When the chest is examined, there may be dullness in the left thorax, tympany on percussion, and the auscultation of adventitious sounds. A succussion splash is heard occasionally, and there is a shift in cardiac dullness and apex beat. When these facts are known, an emergency chest plate is ordered, and the following typical findings are reported:

1. Simulation of a high diaphragm by the herniated viscus.
2. Cardiac shift to the right.
3. Air bubble above the normally anticipated diaphragmatic level (which may be taken for the "magenblase").
4. Fluid in the left chest.

A chest tap, if done, will yield sanguineous or serosanguineous fluid—rarely gastrointestinal contents.

In summary, one finds a patient, generally a man with a history either of trauma or known diaphragmatic hernia, seized with acute left upper quadrant and/or lower left thoracic pain, perhaps radiating to the shoulder, who gives a history of heavy eating, drinking, or physical exertion. He begins to vomit or retch almost immediately, but without significant relief. The findings on physical examination are directed essentially to the chest where there is dullness or tympany of the lower left thorax with suppression of breath sounds and a shift of the heart to the right. Occasionally extraneous bowel sounds may be heard in the chest. If aspiration is done, the contents of the chest may be bloody or serosanguineous. Perhaps gastrointestinal contents may be obtained. Finally, radiography may indicate a portion of the gastrointestinal tract in the chest.

The treatment is obviously surgical. The best approach is through the chest with use of the combined thoracoabdominal incision. This gives better exposure and access to the diaphragmatic hernia, makes the dissection of adherent organs easier, and avoids tears near the heart which are difficult to handle or suture without injury to this organ.

COMMENT

In the case reported in this paper, surgical treatment was obviously impossible because of the fulminating character of the final episode. This was a patient with known diaphragmatic hernia, of presumably congenital origin, discovered on a routine chest film. It is reasonable to assume that on two previous occasions she had gastrointestinal symptoms suggestive of hernial incarceration, with eventual self-reduction of the hernia under conservative therapy.

While it is a well-known fact that uncomplicated diaphragmatic hernia is compatible with long life and may not necessarily require surgical correction, the clinician must be constantly on guard against the surgical emergency produced by strangulation. Furthermore, the repetition or persistence of symptoms in the presence of a known diaphragmatic hernia should lead the clinician to weigh seriously the necessity for surgical correction before an emergency arises.

It is an interesting point that the patient under discussion suffered from cerebral palsy (Little's disease). Perusal of several well-known texts on neurology failed to disclose any relationship between this condition and diaphragmatic hernia.

SUMMARY AND CONCLUSIONS

A case of strangulated diaphragmatic hernia with gangrene of the stomach and perforation into chest and abdomen is presented.

The clinical picture of strangulated diaphragmatic hernia is discussed and an attempt made to indicate the essential points in the diagnosis.

Early diagnosis is the important feature for successful therapy.

Therapy is surgical.

Mr. Herbert A. Fischler, medical photographer at the Jewish Sanitarium, aided in the preparation of the photographs.

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PERSISTENT APPENDICEAL-SIGMOID FISTULA

W. ANDREW DALE, M.D.
ROCHESTER, N. Y.

DESPITE the frequency of acute appendicitis, persistence of a patent fistula between the appendix and another viscus is rare. Recently, a fistula between appendix and sigmoid colon, which had been present for many years, was resected. In this instance, preoperative barium enema diagnosis permitted proper preparation and surgical removal of the fistula. Recognition of the patent tract, particularly at operation for acute appendicitis, would probably have been difficult and might have led to postoperative complications. It therefore appears desirable to report this lesion.

Similar instances of fistula between appendix and other viscera have been reported. These include two cases of appendiceal fistula opening into ileum,[4] and one each between appendix and Meckel's diverticulum,[2] and appendix and ascending colon.[3] In each there was history indicative of acute appendicitis previously. Garcia reported an appendiceal-ileal fistula,[1] without history indicating previous appendicitis. The muscle layer of the appendix and ileum appeared continuous, and Garcia postulated congenital origin in that case. Treatment of these cases consisted of closure of the involved viscera and resection of the appendix. The patient reported on by Gile and MacCarty also had small bowel obstruction due to herniation behind the band formed by the appendix.

In each instance except one it seems likely that the appendix became adherent to the viscus either before or after its perforation due to appendicitis. Spontaneous drainage of appendiceal abscess into bowel is not rare, but persistence of fistula at the site is unusual.

REPORT OF A CASE

M. W., SMH 319295, a 20-year-old white female student, had drainage of appendiceal abscess without appendectomy at age of 2. For several years there had been occasional right lower quadrant pain, and in December, 1952, a severe episode lasted several days. Appendectomy was advised elsewhere but refused.

She then sought further advice here regarding the recurrent abdominal pain. The past history was not remarkable except for moderately severe dysmenorrhea. Positive physical findings were limited to the abdomen where a right paramedian scar was well healed with a central area of depression indicating the site of drainage through the wound. There was slight underlying tenderness. No mass was palpable. Barium enema on two occasions demonstrated a normal colon except for a retrograde passage of barium between the sigmoid and cecum (Figure). This fistulous connection had the appearance of appendix, and after the second barium enema the definite diagnosis was made. Intravenous pyelogram and sigmoidoscopy were negative. Blood cultures, urinalysis, and stool examinations were unremarkable. In preparation for elective resection of the fistula, the patient was started on a bowel cleansing regimen five days prior to surgery. This consisted of a low-residue diet and only liquids for the last 24 hours, administration of 2 gm. phthalylsulfathiazole (Sulfathalidine) four times daily, and a cathartic daily. A Harris tube was passed the preoperative night. At operation, on April 3, 1953, the

From the Department of Surgery, University of Rochester School of Medicine and Dentistry.

pelvis was entered through a low midline incision. Numerous adhesions in the right lower quadrant were cleared to expose the cecum and the base of the appendix. It was difficult to trace the appendix distally where pelvic adhesions fixed the sigmoid colon and attached it to the uterus and posterior parietal peritoneum. After lysis of adhesions the appendix was found attached to the sigmoid almost at its tip. It was cut from the cecum with the phenolized knife and the cecum inverted beneath a double purse-string nonabsorbable surgical (silk) suture. The distal attachment of the appendiceal fistula to the sigmoid was then resected beyond a curved Kocher clamp, while a continuous chromic 00 absorbable surgical (gut) suture was placed over and over in the sigmoid. This was turned in by a layer of interrupted 000 silk sutures. The sigmoid lumen was adequate. Prior to closure a lemon-sized serous cyst of the left ovary was resected, leaving a portion of the ovary in situ. The postoperative course was uneventful, and the patient was discharged after 10 days.

Roentgenogram showing appearance of colon after barium enema.

COMMENT

This fistula between appendix and sigmoid colon apparently formed and persisted following acute appendicitis with perforation and surgical drainage of abscess 18 years prior to further symptoms. Three months prior to hospitalization here, the patient had evidence of acute appendicitis elsewhere but refused advice for operation. After the acute episode spontaneously subsided, she was studied for the cause of persistent mild right lower quadrant symptoms. Barium enema at once indicated an abnormal communication between sigmoid colon and cecum which was confirmed by repeated x-ray study. There was then no difficulty in resecting the appendix after considerable dissection of dense adhesions. Operation during the acute phase through a smaller and less well-placed incision would undoubtedly

have resulted in a much more difficult operation. Blunt dissection of the tip of the appendix from the sigmoid colon while the organ was acutely inflamed could easily have torn away the tip, leaving an opening into the sigmoid colon.

SUMMARY

A case of appendiceal-sigmoidal fistula persistent over 18 years is presented, with comments upon the importance of recognition of this rare lesion whenever the appendix is adherent to another organ.

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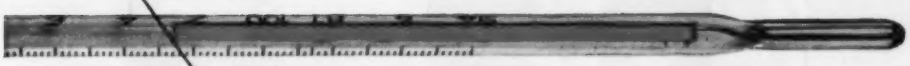
Special Notice

INDEXES

This issue of the ARCHIVES appears in two parts, the second of which is the index for the preceding volume. To make the index more usable to the readers and to enable them to find information with less difficulty than has been encountered in previous volumes, the index has been broken down into three separate indexes: Title Index, Author Index, and Subject Index. The Title Index, which will take the place of the six-month Table of Contents, previously published with the index of the journals, has been prepared in such form that it can be separated from the other indexes and bound in the front of the volume. It consists of the titles of the articles arranged in alphabetical order and accompanied by the name of the author, the name of the senior author if there are more than two, the names of both authors if there are two, and the page number on which the article begins. The Author Index contains the names of all authors of articles appearing in the ARCHIVES and of the presenters of cases or papers before societies. The third index, the Subject Index, contains in alphabetical order the important subjects which have been dealt with in the volume. Individual abstracts are no longer indexed; only the categories under which they have been classified are listed. Book Notices and news items are included in this Index.

when infection strikes...



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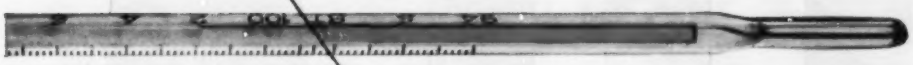

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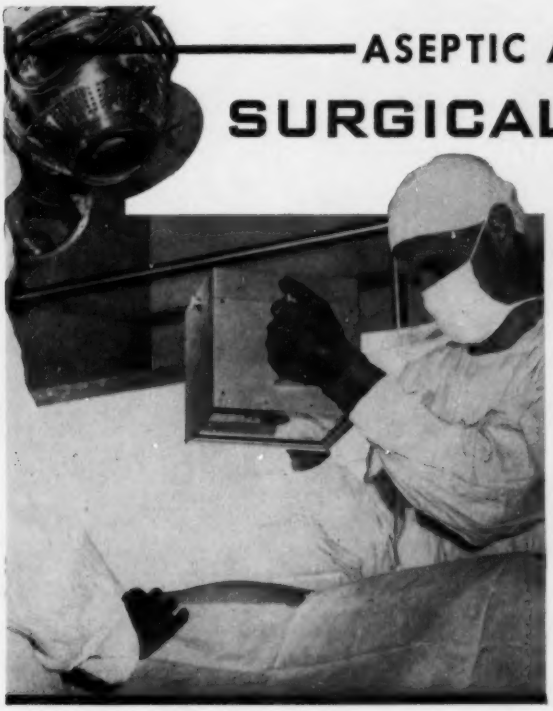

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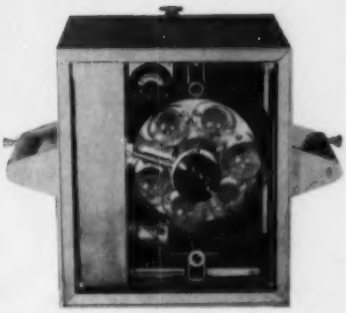

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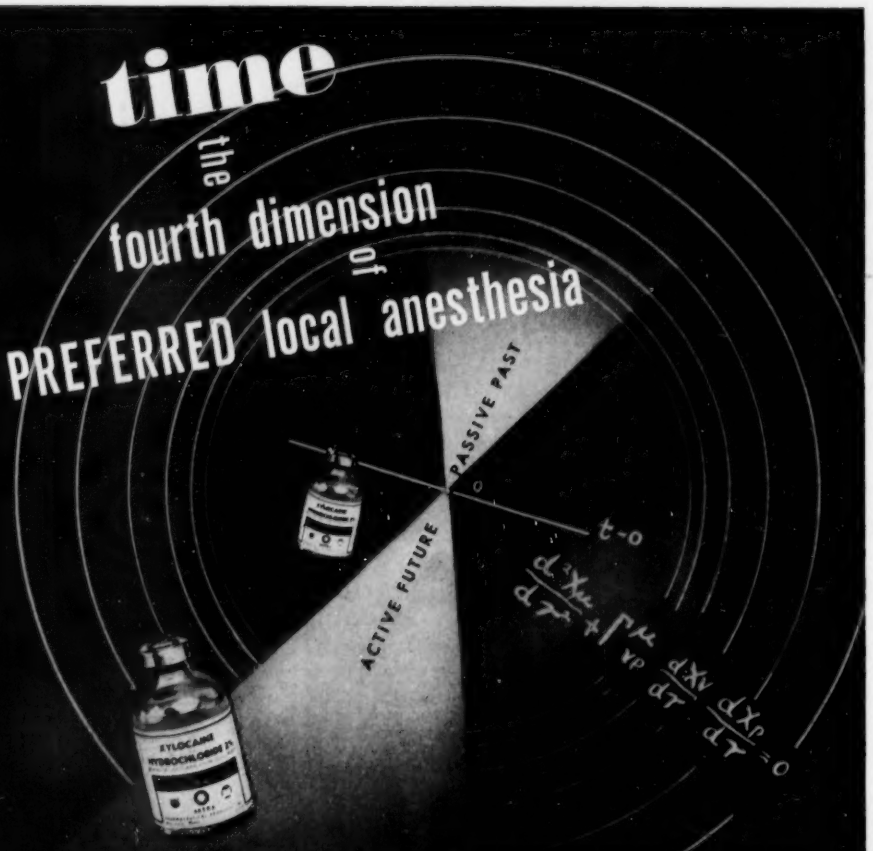

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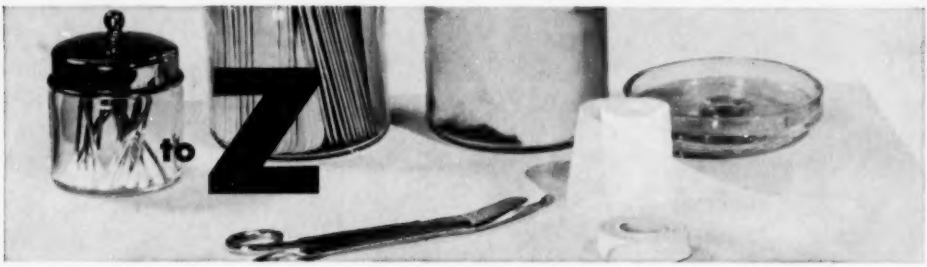

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